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PSYCHOSES ASSOCIATED WITH GROSS BRAIN LESIONS.[1]

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ALTHOUGH I appear before you tonight allegedly to read a paper, I do so, not as an individual, but as the representative of a team consisting of Dr. J. Bentley, Dr. E. J. T. Thompson and myself. Our adoption of team work is no mere submission to fashion, but is the result of the belief that our combined attention to the problems has considerably enhanced the value of the conclusions.

There is a prevalent popular opinion amongst the laity that a piece of bone pressing on the brain is a common cause of insanity. Relatives of patients frequently request X ray examinations of the skull in quite unsuitable cases and they are more than disappointed when they learn that such examinations are of value only in a small proportion of cases. Again, it is very common for relatives to submit a history of a fall on the head as the cause of typical psychoses, like *dementia præcox* or general paralysis of the insane. Although the holders of these popular views are clearly misinformed, it is as well for us once in a while to consider those psychoses which are definitely attributable to gross brain lesions. In so doing, it is necessary to distinguish between mental disorder wholly due to a damaged brain and another group of disorders in which gross brain lesions are detected in mental hospital patients, either clinically after the patient has been in for some time or at *post mortem* examinations, although on admission the patients showed no evidence of brain damage. In these cases it is difficult to say: (i) Whether the brain lesion, while still undetected, was the cause of the mental symptoms; (ii) whether the lesion is only contributory to a preexisting mental disorder or (iii) whether the mental disorder is quite independent of whatever brain lesions may be present. The following recent cases illustrate these points.

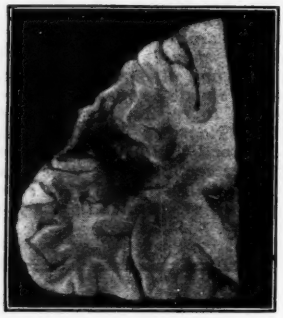

FIGURE I.
Softening in the right occipital lobe.

M.J.S., a female patient, aged fifty-six years, was admitted on October 31, 1929. At that time she was excited, noisy and troublesome. She had delusions of impending harm, for example, that she was going to be buried in quicklime, and in consequence she was depressed and agitated. She showed evidence of hallucinosis. Physical examination revealed no gross lesions. The pupils reacted to light and accommodation. The deep reflexes were feeble, but present, plantar responses were flexor. Her condition was diagnosed as acute melancholia. The condition remained unchanged until rather more than a month after her admission when, while out in the garden, she was noticed to be ill. She became feeble, stupid, her limbs flaccid and her deep reflexes very sluggish or absent. Pulmonary congestion developed and the patient died on December 10, 1929, six days after the onset of feebleness.

Autopsy revealed a softening (not a recent one) near the posterior pole of the right occipital lobe (see Figure I) together with terminal broncho-pneumonia, pronounced atheroma of the cerebral and other vessels and fatty degeneration of the heart.

In this case, in view of the short duration of the illness, about two months, and the presence of a softening of at least a similar duration, it is possible that the brain damage, at first undetected, was the cause of the mental breakdown.

C.D., a male patient, aged thirty-seven years, was admitted on May 2, 1929. He had previously been in an asylum for the blind. He had been several times in Perth Hospital following alcoholic bouts, on several occasions being picked up in the street unconscious. He was admitted from prison.

On examination the heart, lungs and abdomen were found to be normal. The knee jerks were not elicited. The plantar reflexes were flexor in type. No ankle clonus was present. The patient had double glaucoma and gave a history of gonorrhœa and soft chancre. The Wassermann test yielded no reaction. The patient was rambling and disconnected mentally. He was irrelevant in his replies. He had acute hallucinations of hearing, taste and smell with delusions of persecution and grandeur.

A diagnosis of paraphrenia was made. He was very noisy, abusive and excitable. He used neologisms and quoted numbers and became quite incoherent in conversation. He was found dead in bed in the morning of January 17, 1930.

At *post mortem* examination cerebral softening with rupture of the lateral ventricle (through a softening secondary to an old fracture of the skull) was found (see Figure II).

FIGURE II.
Rupture of lateral ventricle.

In this case the brain damage may readily have contributed to the mental disorder.

J.S., a male, aged forty, was admitted on April 15, 1912. According to his family history two brothers and one sister were insane. There was an indefinite history of injury to the skull from a kick by a horse.

On physical examination signs of cardio-vascular degeneration were present. The knee jerks were diminished. The pupils were sluggish. Tremors of the tongue were present. The mental state during his period in hospital was one of recurrent attacks of mania and melancholia with intervening periods when he was employable, but showed signs of dementia. He died of enteric fever on February 24, 1930.

Post mortem examination revealed an old depressed fracture of the vertex of the skull with damage to the underlying brain cortex (see Figure III).

[1] Read at a meeting of the Western Australian Branch of the British Medical Association on March 23, 1930.

The definite family history and the characteristic manic depressive nature of the psychosis make it difficult to associate the mental disorder with the cortical damage, except as a possible exciting cause to precipitate the attack.

At first it may be thought that before certifying any patient, a thorough neurological examination should be undertaken. Dr. Risien Russel has on several occasions maintained in the witness box that such an examination, including ophthalmoscopy, is necessary before certification. However, on more mature consideration it can readily be seen that it is quite immaterial to the certifying medical practitioner whether the person is insane through cerebral softenings or whether the insanity is psychogenic in origin. The alienist, when certifying, is only required to show that by reason of mental disorder the person is unable to look after himself or his affairs and/or is a danger to others. Digression on matters of physical disease is likely to detract from the value of the certificate. For example, when in England, I saw a certificate which stated: "He is suffering from general paralysis of the insane, the Wassermann is positive in blood and fluid. He is confused and dependent." The patient was definitely insane and the diagnosis correct, yet the certificate was valueless and had to be amended, so causing considerable inconvenience to everybody concerned. As an instance of the inadvisability of confusing the mental with the physical examinations, it may be pointed out that certain early general paralytics are not certifiably insane. Actually it may be said that comments with regard to physical disease, no matter how important, are better not included in the certificate proper, but should be stated on the information paper or in a separate letter.

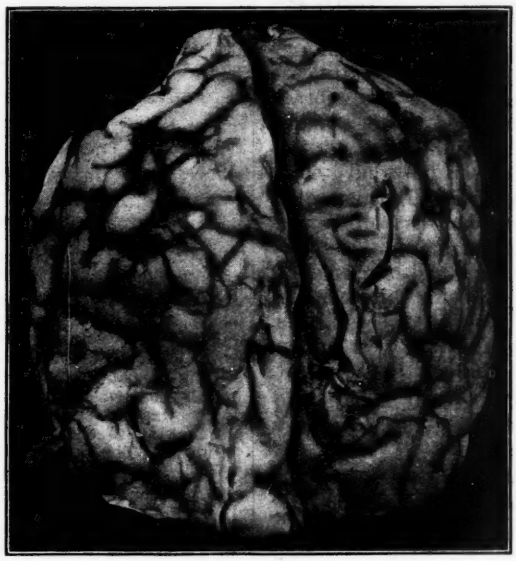

FIGURE III.
Cortex damaged by old fracture of the vertex.

The foregoing does not imply that a physical examination is always unnecessary when dealing with psychotics, as the following example will show. A man may be arrested on the charge of murdering his wife. His mental condition may be questioned and he may then be examined by an alienist whose investigation of the mental state perhaps reveals very little abnormality; the man may be rational, his memory good and he may betray no delusions, hallucinations or other gross mental symptoms. In such a case the alienist would be hard put to it to decide that the man was not responsible for his action. If, however, it is discovered on neurological examination that the man exhibits Parkinsonian symptoms, the alienist is in a strong position when he reports that the criminal act resulted from mental disorder due to a gross brain lesion.

In spite of the diversity of brain lesions which are capable of causing insanity, the mental symptoms they produce possess certain common factors. Mental examination alone is sometimes sufficient to suggest the presence of a brain lesion. The cerebral conditions likely to be associated with psychoses are tumours; softening, scleroses and hæmorrhage, all commonly due to atheroma of the cerebral vessels; traumatic conditions and inflammatory processes, such as encephalitis and even general paralysis of the insane. While typical patients with general paralysis of the insane present a definite mental picture—too well known to merit repetition—there are certain atypical sufferers of whom one can only say in the early stages that they appear to have insanity with some brain lesion. The mental symptoms commonly found in insanity due to organic brain disease are by no means restricted to this condition and they may be found in other psychoses, but nevertheless they occur with sufficient constancy to make them undoubtedly useful in the provisional diagnosis.

One of the symptoms frequently present in gross brain lesions is confusion, associated with defective memory, disorientation and dementia. Other symptoms encountered are persecutory ideas not fully developed into delusions, linked perhaps with obsessions into which the patient has some insight. Thus the patient may think that the medical and nursing attention he is receiving is intended to annoy him and he may then adopt obstructionist tactics, although he really knows that his treatment should benefit him. This is all due to an intense irritability very commonly present in organic brain lesions. One of the patients whom we intend to show (Case II) exhibits this markedly. When asked to answer some simple question or to perform some easy movement, he is liable to express his annoyance in explosive form, although actually he is anxious to assist in the tests.

Emotional instability is very commonly found in association with gross brain lesions. The patients

are sometimes depressed, sometimes elated, their mood being altered by the smallest ordinary daily happening and they are upset by any change in their environment. Moreover, they show disorders of conduct as a result of this lack of emotional balance. Thus patient number I, on realizing his sexual impotence, has attacked his wife, damaged the furniture, abused his children and behaved in a thoroughly objectionable manner. Patient II, when "crossed" in any way, becomes quite violent or feels a strong impulse to do violence. On one occasion this patient for some paltry reason smashed up his furniture with an axe and was only just prevented from battering down the door. At another time he forcibly ejected three men from a union meeting and then sat out on the road laughing loudly. These actions are not like the errors of conduct displayed by patients with delusional insanity, nor are they like the typical outbursts of *dementia præcox* in which there is generally hallucinatory influence and marked dissociation. Typically, the antisocial conduct of psychotics with gross brain lesions results from uncontrolled responses to ordinary upsetting or annoying influences.

Of the two patients to be shown both have some confusion and dementia, both have faulty memories, both display considerable emotional instability with periods of depression and occasional outbursts of an antisocial character, both have had obsessions—Patient I an obsession for exposing himself and Patient II claustrophobia, a fear of closed spaces, while patient II has had in addition persecutory ideas, for example, that he is being followed about.

After this brief description of their mental pictures, we can now consider the cases from a neurological standpoint.

CASE I.—A.E.E. is aged fifty-one years. He was born in England where he worked in the metal foundry at Woolwich Arsenal. His family history revealed no significant nervous disease, his father died of cancer when sixty-four, his mother of pneumonia when fifty-four. Four brothers and five sisters show no evidence of nervous disorder.

In 1913 he came to Australia and enjoyed good health until 1915, when he enlisted and subsequently served nearly three years overseas. During military service he suffered from slight pleurisy, gonorrhœa and an accidental compound fracture of the right tibia. This later condition required prolonged treatment and it was the cause of his discharge from the Army in 1918. Since then he has been unemployed and has complained from time to time of pain and weakness of the left leg, weakness of the right leg, severe pain in the neck and in the sacral region, "rheumatism" of the upper limbs, dyspnœa, general weakness, irritability and depression. In addition his medical history, compiled by the Repatriation Department, shows that he has been regarded in turn as suffering from rheumatism, neurasthenia, valvular disease of the heart and lately *dementia præcox*. He has several times indulged in impulsive and destructive outbursts.

On admission to Claremont on January 15, 1930, his condition was diagnosed as secondary dementia owing to confusion, defective memory and disorientation. He was emotionally unstable, facile and largely lacking in insight.

Physical examination revealed the presence of mitral stenosis, presystolic and systolic bruits being present in the mitral area, a local thrill together with no aortic bruits. There was also slight anæmia. Tendon reflexes were very brisk, particularly so in the lower limbs in which marked hypotonus was present (see Figures IV and V). The arms

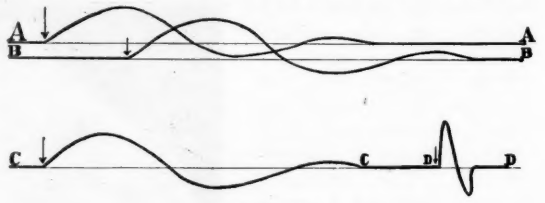

FIGURE IV.

Tracings of knee jerk. Normal controls. A, B and C with drum revolving medium pace and D with drum revolving slowly.

showed less hypotonus and the neck and facial muscles some rigidity. From time to time all the limbs showed rigidity after voluntary movement. Both plantar responses were flexor, there was no ankle or patella clonus and the abdominal and cremasteric reflexes were present. The pupils reacted to light and accommodation, but convergence was defective. There was slight lateral nystagmus on looking to the left. Looking laterally caused definite blinking. The optic discs showed no signs of atrophy.

The gait was unsteady, he walked with a wide base and he made a poor attempt to walk along a straight line; he placed his feet with toes inwardly directed and he frequently lost his balance. He allowed his left arm to swing freely while his right was held to his side. There was difficulty in turning quickly, especially to the left. When walking blindfolded, he showed a varying tendency to deviate to the right. Rombergism was pronounced. A variable degree of muscular incoordination was shown by the finger to nose test, finger to finger test

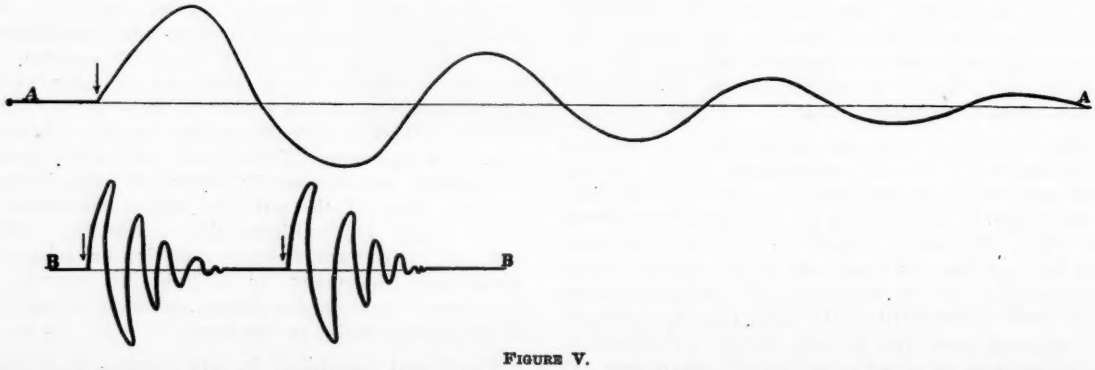

FIGURE V.

Tracings of knee jerk (Case I) showing hypotonus, A with drum revolving medium pace, B with drum revolving slowly.

and the opposing thumb and finger test. He showed no marked dysdiadochokinesis. There were present gross choreiform movements of the head and upper limbs and to a less extent of the lower limbs. These movements were increased with emotion and intention. In addition there were fine athetoid movements of the fingers, particularly marked when the limb was held rigidly. When holding his hands above his head, he held them in the supine position. There was loss of power with pain in the right hand and arm.

The speech was slurred and explosive in character. There were pauses between syllables after which several words were uttered rapidly and in an unnecessarily loud tone. Speech was accompanied by much grimacing. The tongue was shot in and out smartly and symmetrically.

There appeared to be slight loss of sensation, but this was probably due only to his mental condition. The upper dorsal vertebræ showed a slight curvature with the concavity to the right.

There was no loss of sense of position in space and no marked defect in stereognosis. The systolic blood pressure was 122 millimetres of mercury and the diastolic pressure was 84 millimetres. The hæmoglobin value was 75% and the red cells numbered 4,600,000 per cubic millimetre.

In the differential diagnosis one should perhaps consider the following conditions: (i) Functional tics (habit spasm), (ii) disseminated sclerosis, (iii) neurosyphilis, (iv) Huntington's chorea, (v) a cerebellar lesion, (vi) a striatal lesion.

Functional tics, though diagnosed in the early stages, are negatived by the definite physical signs, hypotonus, Rombergism *et cetera*. It would be most unusual for a patient with disseminated sclerosis of twelve years' duration to present no upper motor neurone lesion of the lower limbs (with positive Babinski clonus and spasticity), no changes in the optic discs and no definite nystagmus. The absence of these signs practically excludes disseminated sclerosis. The third condition is disposed of by considering the laboratory reports. No reaction to the Wassermann test was obtained in the blood and cerebro-spinal fluid. There was no increase of globulin or cells in the cerebro-spinal fluid. Lange's gold-sol curve was not abnormal.

The absence of a definite family history, together with hypotonus and other cerebellar symptoms, is sufficient to dispose of Huntington's chorea as a possible diagnosis.

A cerebellar tumour is unlikely owing to the duration of the illness and to the absence of symptoms of increased intracranial pressure. A cerebellar lesion, either a sclerosis or a softening, however, appears to be present. The ataxia, incoordination, hypotonus and dysarthria are all of a typical cerebellar type, while the tendency to deviate to the right when walking blindfolded, the spinal curvature with the concavity to the right, the weakness of the right arm and hand and the fixation of that limb when walking rather indicate a right-sided or a central lesion, though a left-sided lesion might be present in addition. Recent investigations into the functions of the cerebellum throw considerable doubt as to the possibility of exact localization of lesions in that organ.

The choreoathetotic movements, the blinking sign and the rigidity following voluntary movement could be accounted for by a lesion in the *corpus striatum*—a sclerosis or softening in the *putamen*, the caudate nucleus, the optic thalamus, the subthalamic nuclei or the superior cerebellar peduncle. It is worth noting, in view of the coexistence of multilobular hepatic cirrhosis with typical progressive lenticular degeneration (Wilson's disease) that the liver function tests in this case gave normal results.

Multiple lesions which could possibly account for the condition are: (i) Multiple small emboli resulting from the mitral stenosis, (ii) lesions due to early cerebral atheroma, (iii) idiopathic scleroses.

The evidence seems to be in favour of the first of these. On the other hand, a single lesion in the cerebellum, involving also the superior cerebellar peduncle, could perhaps account for all the symptoms.

CASE II.—W.P.T., aged forty years, prior to enlistment in 1915 was an efficient bricklayer. During the war his only recorded illnesses were scabies and acute appendicitis. He states, however, that he was several times blown up and that he fell from a horse in France. Though not on his official record, it is stated that he had influenza when overseas and malaria in Africa in 1920.

In 1919 the patient complained of depression and failing memory and in 1922 he was treated in Perth Hospital for these symptoms and his condition somewhat unaccountably diagnosed as "cerebral concussion." In 1925 he spent a month in Perth Hospital owing to weakness, loss of appetite *et cetera*. While there, he was investigated most thoroughly with a view to detecting pulmonary tuberculosis. Almost throughout his stay in Perth Hospital his temperature varied between 37·2° and 38·9° C. (99° and 102° F.). Physical examination, X ray examination of the chest, a blood count, von Pirquet test and repeated examinations of the sputum showed no evidence of tuberculosis. In 1927, after continued ill health, he developed wrist drop on the right side. The condition was found to be due to a cervical rib which was then resected. Later in the same year he was treated for inability to concentrate, morbid fears, pains in the head, "flushes" of the face and failing memory. At this time no abnormal physical signs were detected in the central nervous system and the optic discs were found to be normal. Further symptoms to develop were slurring of the speech, depression and tremulousness. He indulged in impulsive and destructive outbursts, in some of which he smashed furniture and attacked people without provocation. After these outbursts he displayed emotionalism, laughing foolishly.

The patient was admitted to "Lemnos" Hospital on April 17, 1928, and was there examined. He was regarded as possibly suffering from disseminated sclerosis. He was discharged on April 18, 1929, and readmitted on March 6, 1930.

Physical examination revealed no organic disease of the heart or lungs. The deep reflexes were exaggerated, there was slight ankle clonus on the left side and on that side the plantar response was extensor. On the right side no clonus was obtained and the plantar response was indefinite. The abdominal and cremasteric reflexes were present. Rigidity of the limbs made it difficult to test for clonus. Such was the rigidity that, after a knee jerk, the return of the foot to its resting position was considerably delayed (see Figures IV and VI). The marked rigidity of the legs was associated with hypertrophy of the muscles of the calf and the calf measurement on the left side was 1·25 centimetres (half an inch) greater than that on the right. A slight cogwheel effect was obtained with passive flexion and extension of the arm. There was a fine tremor (increased with excitement and intention) of the head, tongue and hands (see Figure VII). Coordination was poor; this was evidenced by the finger to nose, finger to finger and thumb to finger opposing tests. Speech showed some stuttering with slurring. There were slight internal strabismus, weakness of convergence and a fine

lateral nystagmus. The pupils were equal and reacted to light and accommodation. The discs were normal. There was pigmentation of the iris. He exhibited a degree of Rombergism. Testing of sensation, though difficult owing to imperfect cooperation on the patient's part, revealed no obvious sensory defect. The patient was sluggish in his movements, his face was expressionless, almost mask-like, and his hands and arms were frequently held in a typical Parkinsonian attitude. For a time one observed definite pill-rolling movements in both hands.

Serological tests, repeated on March 15, 1930, revealed no Wassermann reaction in blood or fluid. There was no increase of cells or globulin in the cerebro-spinal fluid. Lange's gold sol curve was quite normal.

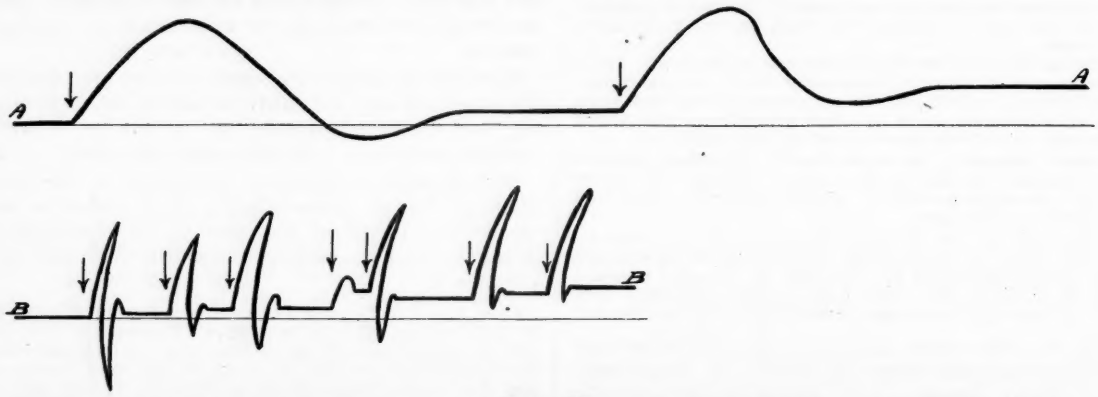

FIGURE VI.
Tracings of knee jerk (Case II) showing hypertonus, A with drum revolving medium pace, B with drum revolving slowly.

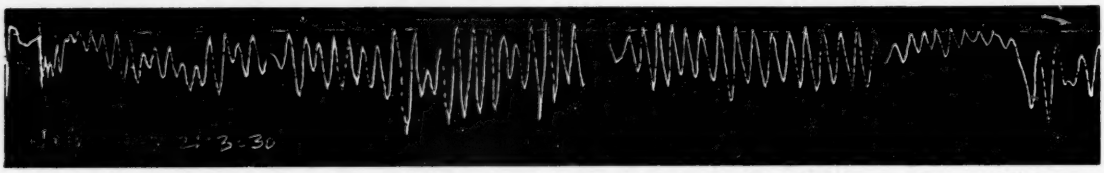

FIGURE VII.
Tracing of intention-tremor (Case II) taken from the extensor surface of the forearm.

It is likely that the condition is one of post-encephalitic Parkinsonism. The upper motor neurone involvement on the left side, the rigidity, the tremor, dysarthria and the ocular signs, together with the characteristic pose, all indicate Parkinsonism. The Parkinsonism might be due to some cause other than *encephalitis lethargica*, namely, *paralysis agitans*. In the former the lesion is accepted as being in the *substantia nigra* and in the latter probably as being in the same region and/or in the large cells of the *corpus striatum*. Damage to these regions might conceivably result from shell concussion sustained when being blown up.

At the same time the indefinite febrile illness in 1925 could easily represent either an attack *de novo* of *encephalitis lethargica* or an exacerbation in the course of chronic encephalitis, possibly starting with influenza in England, but continuing as a progressive disease of the nervous system.

Against disseminated sclerosis, a possible alternative diagnosis, is the characteristic Parkinsonian attitude including "pill rolling," the absence of history of fleeting palsies, the absence of changes in the optic discs and the presence of general rigidity.

Acknowledgements.

In conclusion I should like to thank Dr. Paton for examining the optic discs and Mr. J. L. Larkin for his valuable assistance throughout and particularly for his service in securing tracings of the reflexes.

THE REFRACTIVE INDEX OF THE CEREBRO-SPINAL FLUID USED AS A CHECK ON THE CHEMICAL ANALYSIS.[1]

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In a former paper Penfold and Price,[(1)] after reviewing the literature, brought forward evidence suggesting that the refractive index of the cerebro-spinal fluid provided amongst other things a useful check on the chemical analysis of the fluid. In that paper many of the analyses were very fragmentary, so that the check could not be applied to much of the material dealt with. We therefore determined to collect data of one hundred specimens of fluid

[1] Read at a meeting of the Victorian Branch of the British Medical Association on March 5, 1930.

in which the coagulable protein, chlorine as sodium chloride, urea and glucose were all estimated, to see if by taking cognizance of the refractive effects of the deviations from the normal of all these constituents, we could usefully check the chemical analyses.

The general result of this inquiry has been to show us that in over half the cases dealt with the observed and calculated refractive indices have been in close agreement, but the remaining specimens of fluid have shown such discrepancies between the two indices that we are forced to the conclusion that other constituents vary considerably in concentration and must also be estimated.

Technique.

The refractive index was determined by means of the dipping refractometer of Zeiss, used as described in the before-mentioned paper.[1] The refractometric reading was converted into the actual index by reference to Table I. The various chemical constituents were estimated as follows.

TABLE I.

To enable the Ready Conversion of Scale Readings of the Zeiss Dipping Refractometer into Actual Refractive Indices.

Scale Divisions.	Refractive Index.	Scale Divisions.	Refractive Index.
19·0	1·33474	21·6	1·33574
19·1	1·33478	21·7	1·33578
19·2	1·33482	21·8	1·33582
19·3	1·33486	21·9	1·33586
19·4	1·33490	22·0	1·33590
19·5	1·33494	22·1	1·33594
19·6	1·33497	22·2	1·33598
19·7	1·33501	22·3	1·33601
19·8	1·33505	22·4	1·33605
19·9	1·33509	22·5	1·33609
20·0	1·33513	22·6	1·33613
20·1	1·33517	22·7	1·33617
20·2	1·33521	22·8	1·33620
20·3	1·33524	22·9	1·33624
20·4	1·33528	23·0	1·33628
20·5	1·33532	23·1	1·33632
20·6	1·33536	23·2	1·33636
20·7	1·33540	23·3	1·33640
20·8	1·33543	23·4	1·33644
20·9	1·33547	23·5	1·336475
21·0	1·33551	24·0	1·33667
21·1	1·33555	25·0	1·33705
21·2	1·33559	26·0	1·33743
21·3	1·33563	27·0	1·33781
21·4	1·33567	28·0	1·33820
21·5	1·335705	29·0	1·33858
		30·0	1·33896

Total protein was estimated by precipitation by means of trichloracetic acid. In Mestrezat's method two cubic centimetres of cerebro-spinal fluid *plus* 0·3 cubic centimetre of trichloracetic acid are brought rapidly to the boil, allowed to stand for twenty to thirty minutes to cool and then compared with a scale of standards, prepared in a similar manner from a fluid of known protein content. If the amount of protein is greater than 0·1%, the highest concentration in the standard scale, an approximate value is obtained by diluting the cerebro-spinal fluid with physiologically normal saline solution before making the estimation. If the amount of protein is very high, 300 milligrammes or more, the estimation is usually checked by the refractometer.

Chloride was estimated by titration with standard silver nitrate solution, one drop of potassium chromate being used as indicator. Two cubic centimetres of cerebro-spinal fluid are used and the strength of the solution is such that the burette reading divided by ten gives the percentage of chloride expressed as sodium chloride.

Glucose was estimated by Maclean's method for blood sugar estimation.

Urea was estimated by Maclean's method for blood urea estimation.

The above methods are reasonably accurate for clinical purposes.

In dealing with each fluid, the algebraical sum of the refractive effects of the chemical deviations from the normal average content of the various constituents was added to the normal index. The index so obtained was called the calculated index and, of course, if the chemical examination had been complete, this index should have agreed closely with the observed index. A large number of discrepancies between these indices has been found and these discrepancies have been in both directions, sometimes the observed index being the higher and sometimes the calculated index. We have classified the discrepancies as positive and negative, the positive being those in which the calculated index was higher, the negative those in which the same index was lower than the observed.

An attempt has been made in this paper to define the normal index of man by collecting from our hundred specimens of fluid all those which showed a content of coagulable protein of between 10 and 30 milligrammes, sodium chloride of between 725 and 750 milligrammes, sugar of between 45 and 85 milligrammes, urea of between 3 and 40 milligrammes per 100 cubic centimetres of fluid.[2]

Before any fluid could be put into the group of chemically normal specimens of fluid it had to have the quantity of each of the four above-mentioned constituents lying within the range given.

Workers on the cerebro-spinal fluid will not criticize greatly the ranges chosen, except that the lower limits for sugar and urea may be a little too low. This does not vitiate our series, however, for all the specimens with sugar content between 45 and 68 milligrammes per 100 cubic centimetres were excluded from the chemically normal group by virtue of their marked deviations from the normal in chloride and/or protein content, while no specimens of fluid were found in the series with urea less than ten milligrammes per 100 cubic centimetres.

The upper normal limit for sugar that we have adopted, was that laid down by Polonovski and Duhot: Mestrezat and Eskuchen selected lower upper normal limits for sugar, but their views on this subject have not been supported by recent work.

Only nine specimens of fluid out of the hundred examined have had all their measured constituents fall within the normal ranges. From these specimens a new normal refractive index has been obtained

and new average quantities of the important constituents.

The deviation from the normal in the quantity of any constituent has been determined by taking the difference between the observed quantity and the average concentration of that constituent found in chemically normal specimens of fluid (see Group I). The refractive effects of these deviations have been obtained by consulting Table II.

TABLE II

To show the Refractive Effects of the More Important Constituents of the Cerebro-Spinal Fluid.

Salt and Protein.		Glucose.		Urea.	
Milligrammes.	Units in Fifth Place.	Milligrammes.	Units in Fifth Place.	Milligrammes.	Units in Fifth Place.
10 =	1·75	10 =	1·38	10 =	1·43
20 =	3·5	20 =	2·75	20 =	2·85
30 =	5·25	30 =	4·13	30 =	4·28
40 =	7·0	40 =	5·5	40 =	5·7
50 =	8·75	50 =	6·88	50 =	7·13
60 =	10·5	60 =	8·25	60 =	8·56
70 =	12·25	70 =	9·63	70 =	9·98
80 =	14·0	80 =	11·0	80 =	11·41
90 =	15·75	90 =	12·38	90 =	12·83
100 =	17·5	100 =	13·75	100 =	14·26
200 =	35·0	200 =	27·50	200 =	28·52
				300 =	42·78
				400 =	57·04

The quantities given in the table, when dissolved in one hundred cubic centimetres of cerebro-spinal fluid, produce the refractive effects mentioned.

In Group I the findings are set out as obtained from the examination of specimens of fluid chemically normal according to the before-mentioned standards. These standards differ somewhat in the case of glucose and urea from what were used in the former paper where undue weight was given to certain Continental results, but recent work and our own experience showed this slight change of standards to be desirable.

The most striking result noticed in Group I is that, though a range of 45 to 85 milligrammes of glucose was provisionally regarded as normal in selecting our chemically normal cerebro-spinal fluids, the average obtained from these fluids was actually 76 milligrammes and no fluid in the series had less than 68 milligrammes. We suggest, therefore, that 65 milligrammes should be looked upon as the lower limit of the normal for glucose. Mestrezat believed 48 to 58 milligrammes of glucose to be the normal quantity, Polonovski, on the other hand, 45 to 85. We have found the low readings only with cases of meningitis and naturally they were definitely excluded from the normals because of the marked alterations in the content of other constituents. Polonovski's view on the upper normal limit for glucose is undoubtedly more correct than Mestrezat's.

The average observed index in the nine chemically normal fluids was 1·335093, in close agreement with the normal index that we gave in our original paper, namely 1·33510. The maximum deviation of the refractive index of any single fluid of this group from the average for the whole group was only four units in the fifth place of decimals. The average content for each constituent given in Group I was adopted as our normal standard for use throughout this paper and the deviations found in specimens of fluid in cases of disease were measured from these averages. By way of illustration we will take the fluid from patient number 27, the analysis of which is given in Group III.

The observed values of coagulable protein, sodium chloride, glucose and urea were respectively 240, 580, 40, 15 milligrammes per hundred cubic centimetres. The normal values of these (see Group I) were 24, 732, 76, 24 milligrammes per hundred cubic centimetres. The difference in milligrammes per hundred cubic centimetres was thus +216, −152, −36, −9.

Refractive effects in units of the fifth place of decimals as taken from Table II:

Deviation for protein: +216 milligrammes per hundred cubic centimetres which gives a refractive effect of +37·8 units in the fifth place of decimals.

Deviation for sodium chloride: −152 milligrammes per 100 cubic centimetres which gives a refractive effect of −26·6 units in the fifth place of decimals.

Deviation for glucose: −36 milligrammes per 100 cubic centimetres which gives a refractive effect of −4·95 units in the fifth place of decimals.

Deviation for urea: −9 milligrammes per 100 cubic centimetres which gives a refractive effect of −1·28 units in the fifth place of decimals.

Sum of the refractive effects of the total minus influences = 32·84.

+37·80
−32·84

Difference = 4·96

4·96 units in fifth place +1·335093 (average index of normals) = 1·33514.

1·33514 − 1·33513 (observed index) = difference of 1 unit in the fifth place of decimals.

This shows close agreement between the two indices.

If the refractive influences of these chemical deviations are all added together, the sign of the deviation being disregarded, their sum equals 70·6 units in the fifth place of decimals, so that the check in the case of this particular fluid is extremely good.

In Group I the amounts of the four constituents measured and the refractive indices observed are recorded. The calculated indices are also given and the average difference between the two is only 2·1 units in the fifth place of decimals, while the extreme difference between the two sets of indices is only equal to five units in the fifth place. This shows that no large differences in chemical composition between the various fluids examined occurred in respect of those constituents which had not been estimated. If, for example, a very high concentration of an unestimated constituent occurred in one of the specimens of fluid, then the observed index should have been correspondingly higher than the calculated.

The lower section of Group I deals with four fluids which had their measured constituents within the limits adopted, except that the chlorine was slightly low, namely, between 700 and 725. All these specimens showed the calculated index to be substantially lower than the observed index, so

TABLE III.

Group I: Chemically Normal Specimens of Fluid with Their Indices.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A5	Acute appendicitis	–	30	730	69	25	1·33509	1·33511	– 2
A17	Neurasthenia	–	30	730	68	14	1·33507	1·33507	0
A20	From spinal anæsthesia	–	30	730	68	24	1·33509	1·33505	+ 4
49	Bilateral optic atrophy	–	30	730	83	21	1·33511	1·33513	– 2
A1	Peripheral neuritis	–	20	725	78	40	1·33510	1·33505	+ 5
A6	Idiopathic epilepsy	–	20	730	78	15	1·33507	1·33508	– 1
A8	Cystic degeneration of occipital lobe, glioma	Slightly +	30	730	70	28	1·33510	1·33513	– 3
56	Disseminated sclerosis	–	10	740	83	20	1·33508	1·33509	– 1
10	Chronic frontal sinusitis	–	20	740	83	29	1·33512	1·33513	– 1
	AVERAGE		24	732	76	24		1·335093	2·1
	Fluid with Chloride from 700 to 725.								
A18	Pyrexia of unknown origin, ? cholecystitis	Slightly +	20	700	67	33	1·33503	1·33513	–10
A23	Cerebellar pontine tumour	–	20	700	50	18	1·33499	1·33513	–14
A16	Generalized encephalitis	–	30	720	85	23	1·33509	1·33519	–10
63	Subacute combined degeneration	–	20	700	82	40	1·33506	1·33513	– 7

that we may conclude that some constituent which was not estimated, rose in concentration as the chlorine fell. What this constituent was we have not yet been able to determine.

Group II: Urea Retention.

An epitome of the chemical examination of seventeen specimens of fluid from fourteen patients is given in Table IV, Group II, from which it is seen that the urea concentration of the fluid of all the patients was higher than normal. In the case of patient A13 two specimens were examined, one of which fell within normal limits in respect of urea. Eleven of the specimens of fluid had a high salt content, that is, a greater concentration than 750 milligrammes per 100 cubic centimetres. Strange to say, two specimens had a low chloride content, but whether this was indicative of water retention by the kidney or due to some unrecognized complication we are not able to say. Fluid 19 was obtained from a patient with *herpes zoster*. Ten out of the sixteen specimens of fluid examined had a sugar content of over 100 milligrammes and five of the remaining six had more sugar than was accepted by Polonovski as the normal. The protein concentration was raised above the normal in all but three specimens.

A high urea concentration, therefore, in the cerebro-spinal fluid was usually associated with a high salt, protein and sugar content. The observed refractive indices were shown to have all been above the normal except one, that of fluid number 19, one of the two specimens with a low salt content.

When the calculated and observed indices are compared, considerable differences between them are found to be the rule. The differences are of both positive and negative character, but a large preponderance of the negative differences both in size and number was found. The fact that the calculated index is usually lower than the observed, shows that other substances than those measured were accumulating in the cerebro-spinal fluid. In two cases, on

TABLE IV.

Group II: Urea Retention.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.[1]
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A10	Chronic nephritis	–	70	680	91	68	1·33517	1·33528	–11
A12	Acute nephritis (with œdema)	–	20	770	117	66	1·33527	1·33538	–11
A13	(i) Cerebral tumour right frontal lobe and	–	80	740	98	38	1·33526	1·33540	–14
	(ii) Urea retention	–	40	800	111	50	1·33533	1·33543	–10
A25	Renal tuberculosis	–	70	700	218	384	1·33583	1·33617	–34*
11	Chronic nephritis	–	70	770		163[2]	1·33544	1·33555	–11
30	(i) Polycystic kidneys	–	100	810	110	412	1·33596	1·33590	+ 6
	(ii) Uræmia	–	150	780	122	440	1·33606	1·33600	+ 6
	(iii) With Œdema	–	280	770	136	525	1·33640	1·33640	0
33	Cerebral hæmorrhage	–	40	780	106	62	1·33530	1·33540	–10
41	Uræmia	–	120	850	120	247	1·33585	1·33571	+14*
44	Acute nephritis (with œdema)	–	30	800	126	90	1·33539	1·33536	+ 3
69	Arteriosclerosis	–	80	850	95	55	1·33547	1·33534	+13*
77	Arteriosclerosis, cerebral thrombosis, ? uræmia	–	60	790	90	90	1·33537	1·33551	–14
78	Chronic nephritis	–	100	740	79	57	1·33529	1·33530	– 1
19	Meningo-myelo-radiculitis	–	30	690	123	52	1·33514	1·33513	+ 1
A9	Hemiparesis, ? cerebral thrombosis	–	70	750	91	45	1·33526	1·33553	–27*

NOTE.—Two other cases of urea retention are dealt with in Group VII, 'Organic Nervous Diseases." They were both cases of cerebro-spinal syphilis.

[1] Average – error = 12·9.
Average + error = 7.

[2] Taken from blood.

the other hand, numbers 41 and 69, the calculated index was definitely higher, showing that something had fallen in concentration that had not been measured. This may have been the bicarbonate which is, as far as we are aware, the only other constituent of the cerebro-spinal fluid occurring in sufficient amount as to occasion by its fall in concentration such large positive discrepancies between the two indices. In this urea retention group in only four specimens of fluid out of the seventeen was a reasonable agreement between the observed and calculated indices found.

Group III: Meningitis.

All the specimens of fluid in Group III manifested a cellular increase and a rise in protein. All but one, the first sample from patient number 67, showed a fall in chloride. The sugar content averaged 40·25 milligrammes per hundred cubic centimetres, showing a definite lowering as compared with the normal. The urea was within normal limits in all of the nine cases examined and averaged approximately 21 milligrammes as against a normal average of 24. Seven out of the 13 specimens of fluid manifested a difference between the two indices of not more than five units in the fifth place, while only four out of seventeen showed such close agreement in the urea retention group. Six specimens showed greater discrepancies between the indices and five of the discrepancies were negative in character. The relative prevalence of negative discrepancies is similar to the results obtained in the urea retention series and suggests the increase in the fluid of some substance or substances which had not been estimated.

Group IV: Cardio-Vascular Disease Without Marked Urea Retention.

The most general chemical change in the series was a slight rise of protein, though in one exceptional case this rise was extremely pronounced. The chloride was only infrequently abnormal in amount. The sugar was usually only slightly raised above 85 milligrammes (Polonovski's upper normal limit). The agreement between the indices was nearly as satisfactory as in the normal except in the case of fluid number 53. Here the discrepancy was very pronounced and the protein content enormously high. With a very high protein content of the fluid there is a tendency to negative discrepancies. The significance of this has not yet been investigated.

Group V: Epilepsy.

The observed indices have not the constancy of the normal and four out of the six show significant discrepancies between their calculated and observed indices. Two specimens of fluid manifested a very

TABLE V.

Group III: Meningitis.

No.	Type.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A24	Staphylococcal following repeated ventricular punctures	+	90	600	37	18	1·33492	1·33517	−25*
4	Tuberculous	300	540			1·33524	1·33528	− 4
8	Tuberculous	++	200	650	14		1·33517	1·33521	− 4
13	Tuberculous: Ninth day of disease	+	50	705	56	16	1·33505	1·33513	− 8
	Fifteenth day of disease	+	200	630	21		1·33515	1·33513	+ 2
27	Tuberculous	+	240	580	40	15	1·33514	1·33513	+ 1
52	Tuberculous	+	240	630	37	12	1·33522	1·33547	−25*
66	Pneumococcal	+	300	680	43	30	1·33545	1·33555	−10
67	Tuberculous: First day of disease convulsed	+	40	730	34	20	1·33505	1·33501	+ 4
	Sixteenth day of disease	+	120	620	65	27	1·33505	1·33486	+19*
	Twentieth day of disease (Died on twenty-first day)	+	140	600	53	25	1·33503	1·33513	−10
82	Tuberculous	+	140	680	53	24	1·33517	1·33519	− 2
		+	240	605	30	21	1·33518	1·33513	+ 5

TABLE VI.

Group IV: Cardio-vascular Disease without Urea Retention.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
24	Cerebral hæmorrhage, arteriosclerosis	−	40	740	95	35	1·33518	1·33523	− 5
29	Cerebral thrombosis, arteriosclerosis	−	60	760	80	37	1·33523	1·33521	+ 2
31	Arteriosclerosis, cerebral thrombosis	−	60	730	94	23	1·33518	1·33513	+ 5
37	Arteriosclerosis, cerebral thrombosis	−	60	740	96	19	1·33519	1·33513	+ 6*
64	Arteriosclerosis, cerebral thrombosis	−	70	740	140	30	1·33528	1·33521	+ 7*
22	Right hemiparesis	−	100	740	89	34	1·33527	1·33521	+ 6*
53	Cerebral hæmorrhages, hemiparesis	−	1,000	690	94	27	1·33676	1·33705	−29
47	Paraplegia, auricular fibrillation	−	50	750	106	30	1·33522	1·33521	+ 1
60	Left hemiplegia, aortic regurgitation and fibrillation	−	60	720	95	36	1·33518	1·33513	+ 5

TABLE VII.

Group V: Epilepsy.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A6	Idiopathic..	−	20	730	78	15	1·33507	1·33508	− 1
43	Idiopathic..	−	50	710	97	12	1·33511	1·33524	−13
58	Idiopathic: Fluid I	−	30	700	92	18	1·33506	1·33513	− 7
	Fluid II	Slightly +	40	720	108	18	1·33514	1·33501	+13*
74	? Frontal tumour..	−	30	750	115	17	1·33518	1·33513	+ 5
75	Idiopathic..	−	50	730	98	27	1·33517	1·33523	− 6

definite rise in the refractive index and they were the two with the highest protein content, but both showed negative discrepancies between the indices, so that something must have risen in concentration in the specimens of fluid that had not been measured. The third significant negative discrepancy was associated with a relatively low chloride content of 700 milligrammes. This was shown in Group I usually to be associated with a negative discrepancy.

Group VI: Intracranial Tumours.

In the former paper[1] the tumours dealt with fell into two groups, those with normal indices and those with raised indices. All of the latter were associated with a high protein content of the cerebrospinal fluid. In our present series two specimens of fluid, numbers 34, II, and 50, manifested raised protein without raised observed indices. In these cases positive discrepancies between the two indices were present. Two other specimens, numbers 34, I, and 70, also showed significant positive discrepancies between the indices, for which at the moment we can offer no definite explanation. Three very large negative discrepancies were shown in Group VI. In the case of the two specimens numbers A13, I, and A13, II, they were probably due to the same cause as occasioned the negative discrepancies in the case of the urea retention group. As we have seen in Group II, urea retention is associated with a large number and large size of the negative discrepancies. Fluid A23, II, showed a slight lowering of the chloride, which was shown from Table I to be frequently associated with a negative discrepancy between the two indices. These three negative discrepancies, therefore, probably do not depend upon the presence of the tumours, but on accidental complications. This subject requires further investigation.

Group VII.

In Table IX, Group VII, the results of the examinations of fluid from patients with various organic diseases of the central nervous system are set out. Half of the cases showed good agreement between the observed and calculated indices. The largest discrepancy (−34) occurred in a fluid with the highest index that we have ever observed, 1·33888. If the discrepancy be expressed as a percentage error of the total deviation of the index from the normal, then the error does not appear so very large. In case number 57 a similar large negative discrepancy was associated with a very high protein content. This subject will be discussed later.

Group VIII: Various Infections Not Affecting the Central Nervous System.

The low observed index of the fluid from patient number 71, a case of pneumonia, on the second day of the disease is not explained by any of the estimations made. It may be accounted for by a fall in the bicarbonate, but this is problematical. At present we are examining a series of specimens of fluid quantitatively for protein, chlorine, sugar, urea and bicarbonate to see if by bringing the latter substance also into the calculation we can get rid of the

TABLE VIII.

Group VI: Intracranial Tumours.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A23	Cerebellar pontine tumour: I	−	30	720	88	14	1·33508	1·33511	− 3
	II	−	20	700	50	18	1·33499	1·33513	−14*
A24	Tumour on upper surface of pons	−	30	730	89	19	1·33511	1·33513	− 2
34	Left parietal lobe: I	−	140	740	86	16	1·33531	1·33521	+10*
	II	−	50	750	110	27	1·33522	1·33513	+ 9*
45		Slightly +	240	710	100	24	1·33547	1·33555	− 8
50	Right parietal lobe	−	70	730	110	22	1·33521	1·33513	+ 8*
70	Right temporo-parietal lobe	−	120	750	117	20	1·33534	1·33523	+11*
A8	Cystic degeneration of occipital lobe, glioma	Slightly +	30	730	70	28	1·33510	1·33513	− 3
A13	(i) Cerebral tumour right frontal lobe.. .. and	−	80	740	98	38	1·33526	1·33540	−14*
	(ii) Urea retention..	−	40	800	111	50	1·33533	1·33543	−10*

NOTE.—A13 under the "Urea Retention" series showed also cerebral tumour.

TABLE IX.

Group VII: Various Organic Diseases of the Central Nervous System.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A7	General paralysis of the insane, Wassermann "+++"	+	80	740	86	48	1·33525	1·33519	+ 6*
51	? Tabes, blood Wassermann "+++" ..	−	40	720	90	22	1·33512	1·33499	+13*
39	Disseminated sclerosis	−	20	740	93	17	1·33511	1·33501	+10*
56	Disseminated sclerosis	−	10	740	83	20	1·33509	1·33509	0
A16	Generalized encephalitis..	−	30	720	85	23	1·33509	1·33519	−10*
28	Post encephalitis..	−	60	700	84	22	1·33511	1·33513	− 2
A14	? Encephalitis: Right hemianopia	−	50	740		28	1·33516	1·33513	+ 3
	Right hemiparesis	−	50	730	81	30	1·33515	1·33517	− 2
	Right hemianæsthesia ..	−	50	720		27	1·33512	1·33513	− 1
9	Brain abscess	+	120	740	97	32	1·33532	1·33532	0
6	(i) Brain abscess: Fluid I .. .. .. and	−	30	730	90		1·33512	1·33512	0
	(ii) Abscess of lung: Fluid II	−	30	700	134	30	1·33514	1·33513	+ 1
A4	Pernicious anæmia, subacute combined degeneration	−	60	730	61	32	1·33514	1·33513	+ 1
12	Subacute combined degeneration	−	80	740	68	31	1·33520	1·33513	+ 7*
63	Subacute combined degeneration	−	20	700	82	40	1·33506	1·33513	− 7
2	Spinal tumour (lumbar)	Slightly +	2,000	740	59		1·33854	1·33888	−34*
	(cisternal)	−	100	730	79		1·33523	1·33513	+10*
57	Syphilitic, Wassermann "+" and urea retention	Slightly +	528	680	211	53	1·33611	1·33632	−21*

positive discrepancies. The low sugar found in fluid number 46 may have been due to an incipient tuberculous meningitis. A reference to the first fluid of case number 67, Group III, showed a definite fall of sugar before any change in the chloride concentration, though in this latter case a cellular reaction had also occurred which was not present in fluid number 46 under discussion.

Group IX.

In Group IX it might be pointed out that there is perfect agreement between the observed and calculated indices in the case of a diabetic. The clinical estimation of sugar by Maclean's method we believe to be very accurate for this work, for in several cases of uncomplicated diabetes we have had good agreement between the indices.

In a case of chorea there was found to be definite increase of the protein and chloride in the fluid, while the sugar and urea were within normal limits.

In the case of confusional insanity, the low calculated index taken in comparison with the high observed index suggests that some factor increased greatly in the fluid and has not been measured.

In the case also of arthritis of the spine a definite discrepancy between the two indices occurred, suggesting that something has disappeared from the fluid in a substantial quantity which is not revealed by the analysis.

Discussion.

The average index of the nine chemically normal specimens of fluid was 1·335093 and the maximum deviation of the indices of the normal fluids from this was four units. This swing of the normal minimizes the amount of agreement to be obtained between the observed and calculated indices and suggests that it might be advisable to take the samples of the cerebro-spinal fluid under standard conditions. This, of course, is sometimes not possible. An urgent diagnosis may be required, for example, in a suspected meningitis headache and head retraction may demand an immediate lumbar puncture for their relief. Nevertheless when there is no great urgency, more reliable results could probably be obtained if the conditions of taking the sample were standardized.

A specimen of cerebro-spinal fluid, for example, taken while the patient was fasting, might show the upper normal limit of sugar concentration to be very precise and definite and lead to more definite help in the diagnosis of encephalitis or other disease

TABLE X.

Group VIII: Various Infections not affecting the Central Nervous System.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
A19	Pneumonia: Fourth day of disease	−	30	680	114	28	1·33507	1·33513	− 6
5	Pneumonia: Second day of disease	−	10	730	95	24	1·33509	1·33513	− 4
71	Pneumonia: Second day of disease	−	10	715	112	16	1·33508	1·33494	+14*
15	Gonococcal septicæmia	−	30	690	124	18	1·33509	1·33505	+ 4
79	Infective endocarditis	+	70	670	81	32	1·33508	1·33500	+ 8*
10	(i) Chronic frontal	−	20	740	83	29	1·33512	1·33513	− 1
	(ii) Sinusitis	−	40	700	111		1·33511	1·33517	− 6
55	Deviated septum, sinusitis	−	60	730	68	30	1·33515	1·33513	+ 2
46	Tuberculous peritonitis	−	30	730	37	30	1·33506	1·33513	− 7

TABLE XI.

Group IX: Miscellaneous.

No.	Diagnosis.	Cells.	Milligrammes per 100 Cubic Centimetres.				Refractive Index.		Difference as Units in the Fifth Place.
			Protein.	Chloride.	Sugar.	Urea.	Calculated.	Observed.	
38	Confusional psychosis, bronchopneumonia ..	−	70	730	103	10	1·33519	1·33530	−11
21	Hysterical fit	−	40	730	108	22	1·33516	1·33513	+ 3
61	Concussion	−	50	680	134	39	1·33515	1·33513	+ 2
72	Traumatic headache	−	50	740	100	26	1·33519	1·33509	+10*
48	Chorea	−	60	780	63	40	1·33524	1·33524	0
36	Myositis, arthritis of spine	−	130	740	86		1·33531	1·33517	+14*
73	Cocaine poisoning	−	20	730	98	29	1·33511	1·33509	+ 2
32	Diabetes	−	30	720	206	18	1·33525	1·33525	0
17	Mixed endocrine dystrophy	+	70	740	68	28	1·33518	1·33517	+ 1

where a small rise of the sugar of the cerebro-spinal fluid has been claimed to have significance.

Three specimens of fluid dealt with contained very large amounts of coagulable protein, that is to say, from 500 to 2,000 milligrammes per hundred cubic centimetres. These all showed negative discrepancies. Too much significance should not be attached to these discrepancies, for the method of estimating the protein was not adapted to the estimations of such unusually large quantities.

Summary.

1. About one hundred specimens of cerebro-spinal fluid have had their protein, chloride, sugar and urea estimated.

2. Those specimens of fluid in which the amounts of each measured constituent fell within the normal range, were grouped together as chemically normal.

3. The average refractive index of the chemically normal specimens of fluid equalled 1·335093. The average quantity of each constituent present was: Protein, 24 milligrammes per 100 cubic centimetres; chloride, 732 milligrammes per 100 cubic centimetres; sugar, 76 milligrammes per 100 cubic centimetres; urea, 24 milligrammes per 100 cubic centimetres.

4. The lowest quantity of sugar found in these specimens of fluid was 68 milligrammes per 100 cubic centimetres and we suggest 65 milligrammes as the lower normal limit for sugar.

5. The analysis of each specimen was checked by calculating an index on the basis of the chemical findings and the above-mentioned average index of the chemically normal specimens and comparing the index so obtained with the observed index.

6. The calculated and observed indices agreed well in over half the cases.

7. Discrepancies between the two indices were slight in the chemically normal specimens of fluid, suggesting that the specimens which were normal in respect of the four constituents measured did not differ very materially in their content of the unmeasured constituents.

8. The discrepancies between the indices were classed as positive and negative according as to whether the calculated index was higher or lower than the observed.

9. Marked discrepancies between the two indices were found in fluids showing excess urea. The discrepancies were principally of a negative character.

10. The indices agreed more frequently in the case of fluids from meningitics, but negative discrepancies were not infrequent.

11. The fluids from patients with cardio-vascular disease without urea retention showed, with one exception, good agreement between the indices.

12. Short series of specimens of fluid from epileptics, patients with intracranial tumour, various organic nervous diseases and infections are described.

Acknowledgements.

In conclusion we desire to express our indebtedness to Mr. C. A. E. Price who took the actual observations on refractive index recorded in this paper.

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THE POSSIBLE INFLUENCE OF CLIMATE ON THE INCIDENCE OF PEPTIC ULCER IN AUSTRALIA.

By FRANK L. APPERLY, M.A., M.D. (Oxon), D.Sc. (Melbourne).

(From the William MacLeod Research Laboratory, Pathology Department, University of Melbourne.)

IN 1926 Sundstroem[1] who for some years had been investigating tropical physiology at Townsville, Queensland (latitude 19° 15′ south), published some results showing that in tropical climates human individuals develop an alkalosis. This would appear to be due partly to an over-ventilation of the lungs caused probably by an attempt on the part of the body to get rid of more heat by the respiratory passages. This results in a diminution of carbon dioxide in the blood without, however, a corresponding fall in blood alkali. The latter in fact is increased, though the reason is not clear. The alkali therefore remains in relative excess in the blood.

In 1928 Apperly and Semmens[2] showed that the experimental production of alkalosis, however produced, resulted in a considerable diminution of gastric acidity and emptying time, as tested by the fractional test meal. Conversely, acidosis raised gastric acidity and prolonged the emptying time.

From the above it might be expected to follow that people in Queensland would have a lower gastric acidity on the whole than people in cooler climates and indeed we might expect that the average gastric acidity would progressively rise the more southward people live or, put another way, that the incidence of hyperchlorhydria would progressively rise towards the south, having its lowest incidence in north Queensland and its highest in Tasmania.

Now, as high gastric acidity is certainly a factor in the production of peptic ulcer, we might further expect the highest incidence of peptic ulcer to be in Tasmania and the lowest in Queensland, with intermediate figures in Victoria and New South Wales. If this were so, the advantage of sending suitable patients to Queensland for treatment is obvious.

I was encouraged to investigate the matter further by the experience of Dr. Arthur H. Powell, D.S.O., who after five years of medical practice in Tasmania and three years in Queensland, lately stated to me that in his experience peptic ulcer was more common in Tasmania than in Queensland. Furthermore, it is, of course, common knowledge that cold weather induces or at least aggravates the symptoms in duodenal ulcer.

To obtain statistical proof of my deductions is obviously most difficult. By the courtesy of the Government Statistician in each of the Australian States, I was able to compile two of the accompanying tables. The figures of Table I, however, refer to public hospital admissions only and represent the annual average over five-year periods, except in the case of New South Wales where, prior to 1924, ulcers of stomach and duodenum were grouped under the heading "diseases of the stomach" and were therefore not available separately, while after 1926 the table showing diseases of patients admitted to hospitals was discontinued. In this State, therefore, only the three-year period 1924 to 1926 has been included in Table I.

It will be seen from Table I that the incidence of peptic ulcer appears to diminish fairly considerably as we go northwards, being lowest in Queensland and Western Australia and highest in Tasmania. The other States occupy intermediate positions.

Most of the Queensland figures would, of course, come from the Brisbane hospitals (latitude 27° 30′ south) and it occurred to me that the figures from northern Queensland, which extends to latitude 10° 40′ south, would have been especially interesting for contrast with my figures from other States. It was found impossible to obtain these separate figures from official sources. Through the courtesy and help of Dr. L. J. Jarvis Nye, of Brisbane, I was, however, able to obtain some statistics from Mackay, 520 miles north of Brisbane (latitude 21° 10′ south) for contrast with those of Brisbane. These figures are shown in Table II. The results are in line with those already referred to in connexion with Table I.

In general, then, it would appear that the incidence of peptic ulcer is lowest in hot climates and highest in cold climates.

However this may be, it would appear that climate has no effect on those processes which lead to death from peptic ulcer, such as hæmorrhage and perforation. Table III shows that the death rate for ulcer per 100,000 of the population is about the same in all States.

There are, of course, several obvious possible sources of error in the figures presented. No standard of diagnosis has been or could be adopted, but the percentage of diagnostic error is probably roughly the same in all States. Further, a large incidence of another disease confined to one State only would tend to "squeeze out" ulcer patients from hospital beds and thus make the apparent incidence of the latter less. Fortunately, however, we have no "State diseases" sufficiently marked to have this effect. A study of the "Official Year Book of the Commonwealth of Australia" for the years 1920 to 1928, shows that of all deaths in the Commonwealth due to each of the various diseases, the proportions borne by the various States are roughly the same as those between their populations.

A still further source of possible error is the fact that in those States where there are less hospital

TABLE I.

The Incidence of Peptic Ulcer in Australia (Public Hospitals).

State and Year Period.*	Latitude of Capital City.	Total Hospital Beds (Average).	Average Number of Hospital Beds per 1,000 of Population.	Average Total Number per Year of Ulcers in Public Hospitals.			Ulcer Incidence per 1,000 Beds per Year.		
				Gastric.	Duodenal.	Total.	Gastric.	Duodenal.	Total.
Queensland, 1923–27	27° 30′ S.	4,132	4·95	227	87	314	55	21	76
Western Australia, 1924–28 ..	32° 0′ S.	1,901	4·97	55	41	96	29	21	50
New South Wales, 1924–26 ..	34° 0′ S.	7,461	3·28	?	?	820	?	?	110
South Australia, 1923–27 ..	34° 50′ S.	1,609	2·96	109	75	184	68	46	114
Victoria, 1924–28	37° 50′ S.	4,073	2·40	222	145	367	55	35	90
Tasmania, 1923–28	43° 0′ S.	853	4·00	90	25	115	105	30	135

* States are arranged in order of average temperatures of capital cities according to "Official Year Book of the Commonwealth of Australia."

TABLE II.

*Incidence of Peptic Ulcer in Two Queensland Towns.**

Town.	Latitude.	Period of Observation.	Total Admissions.	Total Ulcers.	Number of Ulcers per 10,000 Admissions.
Brisbane General Hospital, 1925–29	27° 30′ S.	Five years	41,874	505	120·6
Mackay District Hospital, 1920–29	21° 10′ S.	Nine and a half years	14,092	63	44·7

* From figures supplied by Dr. L. J. Jarvis Nye, Brisbane.

TABLE III.

Deaths from Peptic Ulcer in Australia (Whole Population).

State and Year Period.	Average Population during Five-Year Period.	Gastric Ulcer.		Duodenal Ulcer.		Gastric and Duodenal Ulcers.	
		Total for Five Years.	Per 100,000 Population.	Total for Five Years.	Per 100,000 Population.	Total for Five Years.	Per 100,000 Population.
Queensland, 1925–29	835,000	155	18·56	54	6·47	209	25·03
Western Australia, 1924–28	382,600	68	17·77	28	7·32	96	25·09
New South Wales, 1924–28	2,276,000	412	18·10	216	9·49	628	27·59
South Australia, 1924–28	544,200	73	13·42	31	5·70	104	19·12
Victoria, 1924–28	1,698,000	297	17·49	150	8·84	447	26·33
Tasmania, 1924–28	213,000	33	15·49	13	6·11	46	21·60

beds per thousand of the population, the patients with more serious diseases would have preference over ulcer patients, so that the proportion of ulcer patients in hospital to those with other diseases might be altered by, say, doubling the beds, in which case the less serious ulcer patients might be admitted in greater proportion. This objection is refuted, however, by the figures shown in Table I. Probably the most serious objection is that in those States, such as Queensland and Western Australia, where the population is more scattered, there would be a much larger proportion of unfilled hospital beds during the year. If a correction could be introduced for this, the figures which give the ulcer incidence per thousand public hospital beds, would be higher in these two States. This correction, however, would hardly, I believe, raise the figures given for the two States named to the level of those of the southern States. The figures given in Table II, where the proportion of ulcers per 10,000 admissions is shown, are also evidence against this possible source of error.

In conclusion we can claim no more than that these figures are suggestive, but worthy of further investigation. This might be possible, first, by the concerted action of all the Branches of the British Medical Association in Australia, by which more reliable figures might be obtained for the whole population, and, second, by the results of experience of sending ulcer patients to Queensland and comparing the results with those in the southern States. I believe that such an investigation would be worth while.

Acknowledgements.

I wish to acknowledge my debt to Professor W. E. Agar, F.R.S., for his help in the presentation of the statistics here shown; to Dr. L. J. Jarvis Nye, of Brisbane, for his work in obtaining statistics from north and south Queensland and to the Government Statisticians of each of the Australian States.

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THE EXCRETION OF LEAD IN THE URINE AND FÆCES OF PATIENTS INJECTED WITH COLLOIDAL LEAD.[1]

By ROLLO K. NEWMAN, B.Sc., A.A.C.I.,
Sydney.

THE excretion of lead in the urine and fæces of several patients receiving intravenous colloidal lead injections was investigated to learn something of the rate at which the injected metal leaves the system. With two exceptions the subjects were in the Royal Prince Alfred Hospital, the authorities of which kindly arranged for the collection of the entire twenty-four hour output from each patient and made it available for analysis. The duration of the investigation in each instance was determined by the period of retention in hospital and this in most instances was rather short; further, the persons concerned did not receive a full course of lead treatment and it is probable that the average rate of excretion found is somewhat lower than that which occurs in patients receiving more frequent injections. These were the only persons available

[1] This work was carried out under the control of the Cancer Research Committee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

for the work, however, and it will be noted further that all are women, there being no men under treatment at the time.

In some instances the stated average volume of urine per week is very low, but inquiries at the hospital confirmed the figures given as the total output. In the first case, that of C.E.E., the very low urine output may be partly accounted for by the fact that large quantities of peritoneal fluid were tapped at frequent intervals.

On arrival of the urine and fæces at the laboratory each day, duplicate samples, each one-eighth of the whole, were taken from both, the usual sampling precautions being observed, and the samples taken each day were added to those preceding, until at the end of a week duplicate samples representative of the weekly output were available for analysis in the case of both urine and fæces. The lead was estimated by the method described by Avery, Hemingway and Anderson.[1] Sometimes when bismuth medication had been administered, estimation of lead in the fæces was impossible.

The urine was always tested each day on arrival for blood and albumin. No trace of blood was ever detected, but albumin was found as follows:

CASE OF C.E.E.:
First injection, 75 milligrammes of lead.
Albumin absent during next 7 days
Albumin present during next 10 days
Albumin absent during next 4 days
Albumin present during next 5 days
Albumin absent during next 9 days
Second injection, 40 milligrammes of lead.
Albumin present during next 8 days
Albumin absent during next 5 days
Albumin present during next 19 days
Albumin absent during next 10 days
Albumin present during next 2 days
Albumin absent during next 3 days
Albumin present during next 5 days
Albumin absent during next 8 days
Third injection, 45 milligrammes of lead.
Albumin absent during next day
Albumin present during next 2 days
Patient discharged from hospital.

CASE OF A.B.:
First injection, 72 milligrammes of lead.
Albumin absent during next day
Albumin present during next 5 days
Albumin absent during next 1 day
Albumin present during next 10 days
Second injection, 86·4 milligrammes of lead.
Albumin present during next 8 days
Albumin not detected thereafter during next 7 weeks
Patient discharged from hospital.

CASE OF M.A.P.:
First injection, 54 milligrammes of lead.
Albumin absent during next day
Albumin present during next 16 days
Second injection, 57·6 milligrammes of lead.
Albumin present during next 5 days
Albumin not detected thereafter during next 3 weeks
Patient discharged from hospital.

CASE OF C.E.D.:
First injection, 80 milligrammes of lead.
Albumin absent during next 9 days
Patient discharged from hospital.

CASE OF N.L.:
First injection, 75 milligrammes of lead.
Albumin absent during next day
Albumin present during next 9 days
Patient discharged from hospital.

CASE OF B.:
First injection, 75 milligrammes of lead.
Albumin present during next 14 days
Second injection, 75 milligrammes of lead.
Albumin present during next 5 days
Patient discharged from hospital.

The appearance of albuminuria after the first injection occurred in one instance on the first day, in three on the second day, in one on the eighth day and in another had not occurred by the ninth day. Once albuminuria appears, it seems likely to recur during periods ranging from five days to several weeks. It is not possible to affirm, however, that lead injections are the cause of the recurrent albuminuria in the case of C.E.E.; in fact it is probable that other causes are more important in this instance. The normal behaviour is probably more accurately indicated by patients A.B. and M.A.P. where eight days and five days respectively after the second injection the albuminuria cleared up permanently.

The results obtained from the analysis of urine and fæces for lead follow:

PATIENT C.E.E.: Sex, female; age, forty-nine years; disease, diffuse sarcoma of peritoneum; period of investigation, March 27, 1928, to July 3, 1928, fourteen weeks.

Lead injected and notes from hospital:
March 27, 1928: 75 milligrammes of lead injected. No note *re* reaction.
April 5, 1928: Red cell count, 4,330,000 per cubic millimetre; leucocytes, 10,400 per cubic millimetre.
April 17, 1928: Red cell count, 4,000,000 per cubic millimetre.
May 1, 1928: 40 milligrammes of lead injected.
May 2, 1928: Does not feel so well after lead injection yesterday, but she is not collapsed. Pulse is quite good.
May 28, 1928: No stippling of red blood cells detected in great numbers of fields examined.
June 30, 1928: 45 milligrammes of lead injected.
July 3, 1928: Distended vomiting, enema with very little result. Patient's condition was not so good. Condition was worse.
July 5, 1928: Patient was no better and condition slightly worse, abdomen very distended, vomiting present.
July 5, 1928: Transferred to "Home of Peace," Marrickville. No further details known.
Total lead injected was 160 milligrammes.

Urine received at laboratory:

Average volume per week ..	3,520 cubic centimetres
Maximum volume in one week	5,865 cubic centimetres
Minimum volume in one week	2,175 cubic centimetres

Lead found in urine:

Average quantity per week	1·33 milligrammes
Maximum quantity in one week ..	2·08 milligrammes
Minimum quantity in one week ..	0·77 milligramme

Lead concentration of urine:
Average concentration, 0·38 milligramme per litre of urine.
Maximum concentration, 0·70 milligramme per litre of urine.
Minimum concentration, 0·19 milligramme per litre of urine.

Fæces received at laboratory:

Average dry weight per week	103 grammes
Maximum weight in one week	176 grammes
Minimum weight in one week	62 grammes

Lead found in fæces:

Average quantity per week	2·50 milligrammes
Maximum quantity in one week ..	4·14 milligrammes
Minimum quantity in one week ..	1·03 milligrammes

PATIENT A.B.: Sex, female; age, twenty-one years; disease, melanotic sarcoma of soft tissues. Period of investigation, May 22, 1928, to August 8, 1928, eleven weeks.

Lead injected and notes from hospital:
May 22, 1928: 72 milligrammes of lead injected (45 milligrammes intravenously, 27 milligrammes into tumour).
May 23, 1928: No ill effects from lead.
May 28, 1928: No red cells showing punctate basophilia.
June 8, 1928: 86·4 milligrammes of lead injected (43·2 milligrammes intravenously, 43·2 milligrammes into tumour). No collapse signs. Patient was recommended for further readmission; was not admitted. No record of weight made. No other blood counts done.
Total lead injected, 158·4 milligrammes.

Urine received at laboratory:
Average volume per week .. 5,183 cubic centimetres
Maximum volume in one week 6,995 cubic centimetres
Minimum volume in one week 2,885 cubic centimetres

Lead found in urine:
Average quantity per week .. .. 2·17 milligrammes
Maximum quantity in one week .. 6·44 milligrammes
Minimum quantity in one week .. 0·80 milligramme

Lead concentration of urine:
Average concentration, 0·42 milligramme per litre of urine.
Maximum concentration, 0·97 milligramme per litre of urine.
Minimum concentration, 0·20 milligramme per litre of urine.

Fæces received at laboratory:
Average dry weight per week .. .. .. 108 grammes
Maximum weight in one week .. .. .. 152 grammes
Minimum weight in one week .. .. .. 69 grammes

Lead found in fæces:
Average quantity per week .. .. 4·43 milligrammes
Maximum quantity in one week .. 11·32 milligrammes
Minimum quantity in one week .. 1·56 milligrammes

PATIENT M.A.P.: Sex, female; age, sixty-three years; disease, sarcoma of right maxilla; period of investigation, May 22, 1928, to July 4, 1928, six weeks.

Lead injected and notes from hospital:
May 22, 1928: 54 milligrammes of lead injected.
May 23, 1928: Very well; no signs of collapse.
May 27, 1928: Patient's condition was excellent. No serious signs due to lead were present.
June 8, 1928: Colloidal lead 57·6 milligrammes injection. No note *re* reaction.
July 5, 1928: Patient discharged for observation. No further record made; no blood count made; no record of weight made. No note when dead.
Total amount of lead injected, 111·6 milligrammes.

Urine received at laboratory:
Average volume per week .. 5,608 cubic centimetres
Maximum volume in one week 6,635 cubic centimetres
Minimum volume in one week 4,725 cubic centimetres

Lead found in urine:
Average quantity per week .. .. 1·77 milligrammes
Maximum quantity in one week .. 2·76 milligrammes
Minimum quantity in one week .. 1·03 milligrammes

Lead concentration of urine:
Average concentration, 0·32 milligramme per litre of urine.
Maximum concentration, 0·42 milligramme per litre of urine.
Minimum concentration, 0·19 milligramme per litre of urine.

Fæces received at laboratory:
Average dry weight per week .. .. .. 83 grammes
Maximum weight in one week .. .. .. 96 grammes
Minimum weight in one week .. .. .. 75 grammes

Lead found in fæces:
Average quantity per week .. .. 2·59 milligrammes
Maximum quantity in one week .. 3·80 milligrammes
Minimum quantity in one week .. 1·36 milligrammes

PATIENT C.E.D.: Sex, female; age, forty-five years; disease, carcinoma of *cervix uteri*; period of investigation, June 12, 1928, to June 19, 1928, one week.

Lead injected: June 12, 1928, 80 milligrammes. No other notes from hospital available.
Urine received at laboratory: 4,053 cubic centimetres.
Lead found in urine: 1·28 milligrammes.
Lead concentration: 0·32 milligramme per litre of urine.
Fæces received at laboratory: 63 grammes (dry weight).
Lead found in fæces: 2·12 milligrammes.

PATIENT N.L.: Sex, female; age, forty-three years; disease, carcinoma of *cervix uteri*; period of investigation, June 30, 1928, to July 6, 1928, one week.

Lead injected: June 30, 1928, 75 milligrammes. No other notes from hospital available.
Urine received at laboratory: 3,945 cubic centimetres.
Lead found in urine: 0·76 milligramme.
Lead concentration: 0·19 milligramme per litre of urine.
No figure for fæces obtainable.

PATIENT B: Sex, female; age, thirty years; disease, secondary sarcoma of lung; period of investigation, May 14, 1928, to June 2, 1928, nineteen days.

Lead injected: May 14, 1928, 75 milligrammes; May 28, 1928, 75 milligrammes.
Urine received at laboratory (in this case not total excretion): 4,520 cubic centimetres during nineteen days.
Lead found in urine: 1·45 milligrammes.
Lead concentration: 0·32 milligramme per cubic centimetre.
Fæces received at laboratory: (Total) 283 grammes dry weight in nineteen days = 104 grammes per week.
Lead found in fæces: 6·20 milligrammes in nineteen days, equivalent to 2·28 milligrammes per week.

PATIENT X: Sex, female; age, sixty-four years; disease, malignant pylorus.

Lead injected: 400 milligrammes between May 6, 1928, and July 29, 1929.
Urine sample, 215 cubic centimetres, taken on August 14, 1929, sixteen days after last injection.
Lead found: 0·07 milligramme in 215 cubic centimetres or 0·325 per litre.
No fæces were examined.

These figures, summarized in Table I, show that the injected lead is only slowly excreted in the urine and fæces.

TABLE I.

Patient.	Total Lead Injected in Milligrammes.	Period of Investigation.	Lead Excreted in Urine.		Lead Excreted in Fæces.		Total Lead Excreted per week per 50 Milligrammes of Lead Injected.	Lead Excretion as Percentage of Lead Injected.
			Total in Milligrammes.	Milligrammes of Lead per Week per 50 Milligrammes of Lead Injected.	Total in Milligrammes.	Milligrammes of Lead per Week per 50 Milligrammes of Lead Injected.		
C. E. E.	160·0	14 weeks	18·6	0·415	35·0	0·78	1·20	2·4
A. B.	158·4	11 weeks	23·9	0·680	48·7	1·40	2·08	4·16
M. A. P.	111·6	6 weeks	10·6	0·800	15·5	1·16	1·96	3·92
C. E. D.	80·0	1 week	1·28	0·800	2·12	1·32	2·12	4·24
N. L.	75·0	1 week	0·76	0·510	—	—	—	—
B.	150·0	2·7 weeks	—	—	6·20	0·77	—	—
			Average ..	0·641	—	1·09	1·84	3·68

It is of interest to compare the excretion of these patients with that of people suffering from lead poisoning. Tannahill[2] finds in the urine of smelter employees exposed to lead absorption a lead concentration of 0·02 to 1·04 milligrammes per litre with an average of 0·27 milligramme per litre. Badham and Taylor[3] find from 0·01 to 0·53 milligramme per litre in the urine of persons certified to be suffering from lead poisoning. The figures given in Table II will be seen to fall between these limits.

The concentration of lead in the urine during the week following the first injection is for the average person low, the mean figure for several patients being 0·21 milligramme per litre. After subsequent injections, however, the concentration during the following week may be considerably increased; thus in the case of C.E.E. the maximum figure of 0·70 milligramme per litre was obtained during the week following the third injection and the figures of 0·97 milligramme per litre and 0·42 milligramme per litre were obtained in patients A.B. and M.A.P. during the week following the second injection. This possibly indicates a decrease in the readiness with which the body is able to remove the injected lead from the circulation and offers an explanation for the observed clinical fact that patients who exhibit no symptoms after the first injection, may frequently do so after subsequent injections.

Between injections the concentration of lead in the urine fluctuates under the influence of factors which have not been investigated in this work, but are probably related to the diet. In the case of A.B. above, in which part of the lead administered was injected intravenously and part injected into the tumours with adrenalin in saline solution as adjuvant, a very much higher rate of excretion was observed than in the other cases. During the first three weeks, in which 158·4 milligrammes of lead were administered in two injections, the average concentration in the urine was 0·95 milligramme per litre and in the eight following weeks when no more lead was injected, the concentration fluctuated between 0·20 and 0·27 milligramme per litre.

Aub and his fellow workers[4] find in those suffering from lead poisoning, but removed for some time from exposure to lead, excretion at rates varying between 0·54 and 1·82 milligrammes per week in the urine and 0·14 and 4·35 milligrammes per week in the fæces, the average ratio between lead output in urine and fæces being 1:2·5. The figures obtained here are shown on this basis in Table III and are somewhat higher, as would be expected, since the subjects of Aub's work had been removed from exposure to lead for some time, but nevertheless they are generally of the same order. The ratio of excretion in urine to that in fæces shows that a slightly greater proportion of the excreted lead is found in the urine of patients injected intravenously and this again is obviously to be expected.

Conclusion.

The results of the examination of the excretion of lead following injections support the belief that the therapeutic action of the lead is a quantitative rather than a qualitative one. At the average rate of excretion found it would take more than ten weeks for the complete excretion of a single injection of fifty milligrammes of lead and for most of that period the lead concentration in the urine and therefore probably in the blood also would be approximately of the same order as that found in

TABLE II.

Showing Average Concentration of Lead in Urine during Treatment.

Patient.	Total Lead Injected in Milligrammes.	Period of Investigation.	Milligrammes of Lead per Litre of Urine.	Maximum for One Week.	Minimum for One Week.
C. E. E.	160·0	14 weeks	0·38	0·70	0·19
A. B.	158·4	11 weeks	0·42	0·97	0·20
M. A. P.	111·6	6 weeks	0·32	0·42	0·19
C. E. D.	80·0	1 week	0·32	—	—
N. L.	75·0	1 week	0·19	—	—
B.	150·0	19 days	0·32	—	—
X.	400·0	One sample 16 days after last injection.	0·33	—	—
		Average	0·33	—	—

TABLE III.[1]

Patient.	Lead in Urine in Milligrammes.			Lead in Fæces in Milligrammes.			Ratio of Urine to Fæces.
	Average per Week.	Maximum.	Minimum.	Average per Week.	Maximum.	Minimum.	
C. E. E.	1·33	2·08	0·77	2·50	4·14	1·03	1:1·88
A. B.	2·17	6·44	0·80	4·43	11·32	1·56	1:2·05
M. A. P.	1·77	2·76	1·03	2·59	3·80	1·36	1:1·47
C. E. D.	1·28	—	—	2·12	—	—	1:1·66
Average	1·64	—	—	2·91	—	—	1:1·77

[1] Case A. B. in which the lead was injected partly into the masses and partly intravenously is again conspicuously different from the average in the above.

cases of lead poisoning. Clinical experience has shown, however, that this concentration is not sufficiently great to produce therapeutic results, since injection of fifty milligrammes at infrequent intervals is not satisfactory; in fact it appears to be necessary to inject at comparatively frequent intervals to secure results. Under these circumstances the average concentration of lead in the urine and in the blood throughout the period of treatment would probably approximate to the maxima in the cases investigated in this work. In fact one is tempted to suggest that the dose and the frequency of injection in each person treated should be determined by reference to the concentration of lead in the urine, lead being administered as necessary to keep this figure at 0·50 to 0·10 milligramme per litre, for as long as the body can tolerate it. It will be noted that in the above there is an implied assumption that it is the lead in the circulation which is significant and it must be admitted that this is only an assumption. One can only state in justification that there is ample evidence to show that cancer growth may continue uninterruptedly in bodies with considerable quantities of lead locked up in the tissues. It is hoped that in the future an opportunity may arise to follow the lead concentration in the urine of a patient successfully treated with lead and observe whether there is evidence of a maintained high figure.

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Reports of Cases.

MULTIPLE INJURIES.[1]

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IN a metropolitan hospital such as Sydney Hospital, one is accustomed to attending many patients with multiple injuries, but the case here recorded is rather out of the ordinary (*a*) in its multiplicity, (*b*) in the varieties of anæsthesia employed, (*c*) in showing the superiority of active movements over passive movements in restoring function to injured joints, (*d*) in the onset of appendicitis during recovery from injuries, (*e*) in the excellent results achieved and (*f*) in the necessity of taking a lateral radiograph of the spine as well as an antero-posterior one. The details are as follows.

[1] Read at a meeting of the Sydney Hospital Clinical Society on March 5, 1930.

J. F., aged thirty-nine years, was admitted under my care to Sydney Hospital on September 21, 1929, having fallen fifty feet off a building; in the casualty room he was having convulsions; he had blood coming from his nose; there was a lacerated wound of the vertex. A fracture of both bones of the right leg in the lower third was apparent; the fragments were almost projecting through the skin. On account of his cerebral injuries lumbar puncture was attempted two days after admission, but was unsuccessful.

About this time a radiograph of the spine revealed a fracture of the bodies of the twelfth dorsal, first and second lumbar vertebræ, in good position; also a fracture of the second, third and fourth transverse processes of the lumbar vertebræ on the right side and of the second transverse process on the left. No fracture of the skull was seen.

On September 26, 1929, because of his restlessness and for fear of the fracture becoming compound, an open operation was performed under spinal anæsthesia ("Stovaine") on the right tibia (the lower fragment of which was displaced anteriorly and medially). The fragments were got into good position and fixed with a Lane's plate; the wound healed up by first intention.

On October 30, 1929, under local anæsthesia the plate and screws were removed. This is my usual custom after about four weeks in dealing with the tibia.

On October 18, 1929, the right wrist received operative attention under general anæsthesia. The radiograph had revealed a fracture of the lower end of the radius, of the ulnar styloid, of the navicular, also a forward dislocation of the lunate bone. The lunate and fragment of the navicular (both of which were dislocated anteriorly) were removed by means of an anterior incision. The wrist was put up in dorsiflexion in plaster of Paris. At the same time the deformity in the left wrist was reduced and put up in plaster. The X rays had shown here a fracture of the lower end of the radius and of the ulnar styloid, together with a fracture dislocation of the navicular dorsally.

Some radial deviation had developed; this was over-corrected and then the wrist put in a position of dorsiflexion and ulnar deviation.

On November 7, 1929, the plaster was taken off both wrists. He was given tennis balls and subsequently golf balls to play with and warned that the subsequent function of his hands rested very largely with himself; this had the desired effect; he never ceased (except during sleep and at meal times) playing with the balls, rolling his fingers around them.

By the end of the year the movements of wrists and fingers were quite good.

On January 11, 1930, he began to complain of dyspeptic symptoms and of pain in the right iliac fossa; there was some tenderness there on palpation.

On January 14, 1930, appendicectomy was performed under narco-local anæsthesia. A hypodermic injection of morphine 0·015 gramme (one quarter of a grain) and of hyoscine 0·43 milligramme (one one-hundred-and-fiftieth of a grain) was given one hour before the operation and the operation area was infiltrated with a 0·5% solution of "Novocain" and adrenalin one in 150,000. The appendix was definitely pathological, being distended in its distal portion and containing stercoliths.

The operation went off quite successfully; at the end of the operation he conversed with us, saying that he had felt practically nothing; he was in a drowsy condition throughout; this I consider to be a most important factor.

Since this operation he has been free of practically all his abdominal trouble; for a few days he had some discomfort in the right hypochondriac region. Thinking he might possibly have some cholecystitis, I had Graham's test performed upon him, but the X ray report was that the gall bladder filled normally.

At the present (February 19, 1930) he is wearing a plaster spinal jacket preparatory to a celluloid one being made by Miss Moore, one of the hospital masseuses, under the supervision of Dr. Nigel Smith.

He left hospital on February 17, 1930, on crutches. The condition of his fractures is excellent; all are in good

position. There is some limitation of dorsi and palmar flexion in the left wrist, but with this excepton, also that the spine shows a slight degree of kyphosis (according to the radiograph taken on February 19, 1930), the fractures are normal.

One cannot help being struck with the different appearances shown by the antero-posterior and lateral radiographs of the spine. The lateral is far more valuable.

As regards the left wrist, I think I would have been wiser to have removed the navicular in order to give him freer wrist movements; it may still be advisable.

He gets attacks of melancholia at times; whether this is the result of his head injury or is due to something preexisting I am unable to say.

Acknowledgements.

I am indebted to Dr. Voss and Dr. Cutler, Honorary Radiographers of the Sydney Hospital, for the X ray reports.

Reviews.

OBSTETRICS.

THE fourth edition of Jellett and Madill's "Manual of Midwifery" is a thoroughly revised work for which Dr. Madill, in the preface, claims that notice has been taken of all important recent developments in obstetrics.[1] The claim is in every sense justified. The result is an up-to-date work covering the whole field of obstetrics. While, as is natural, particular attention has been given to the teachings and practice of the Rotunda Hospital, the theory and practice of other schools have received due consideration.

The opening chapters on anatomy are followed by a full and well illustrated account of the development of the ovum and placenta. Recent research has contributed to make this a more than usually clear and interesting exposition.

The chapter on obstetric diagnosis leaves nothing to be desired. The methods and advantages of abdominal palpation are fully described and the importance of reducing vaginal examination to a minimum is duly emphasized.

In the section dealing with accidental hæmorrhage the ætiology is fully discussed, reference being made to the views of Fitzgibbon, Couvelaire, Williams and Morse, with an inclination to the theoery that toxæmia and albuminuria are the causative factors in the more severe cases.

In the treatment of this form of hæmorrhage the authors recommend plugging the vagina tightly and so compressing the cervix. They do not favour rupturing the membranes in every case, regarding it as permissible only when it is certain that it will be followed by contraction of the uterus on the fœtus, that is, it is permissible only when the patient is in labour. Cæsarean section is advocated in urgent cases where concealed hæmorrhage coexists with external hæmorrhage. In the discussion of unavoidable hæmorrhage the ætiology of *placenta prævia* is summed up as not certainly determined; three possible factors are indicated. Interesting reference is made to that rare cause, cervical placenta. The treatment favoured is a modified form of the bipolar version of Braxton-Hicks, for which Dr. Jellett and Dr. Madill claim certain advantages over the classical method. This method, we think, will appeal on the score of recognizing in the fullest degree and meeting the possibilities of what is by no means an invariably simple obstetrical manœuvre. A number of alternative measures for dealing with unavoidable hæmorrhage are discussed and regarded with disfavour, for example, Champetier de Ribes's bag, *accouchement forcée*, plugging of the vagina, while Cæsarean section as a routine procedure receives special condemnation. In this regard we view with satisfaction the laying down of a definite line of treatment by the authority of experience.

In discussing the management of the third stage of labour, the authors give approval to the Dublin teaching that the placenta should be expressed as soon as ever it has left the uterus and passed into the vagina. On the other hand, "if it does not leave the uterus within an hour or an hour and a quarter, the normal mechanism is considered to have broken down and the placenta is expressed from the uterus."

New and important matter has been incorporated in the chapters on the toxæmias of pregnancy. The section on the ætiology of eclampsia covers a wide range of theories, Young's theory of placental infarction with breaking down of infarcted areas of the placenta being specially referred to.

Additional matter has made the treatment of contracted pelvis more helpful and skiagrams add to the interest. Nearly one hundred and fifty pages are devoted to obstetrical operations and the final chapters are concise and contain a useful account of infant physiology and pathology and the care of the infant generally. The authors are to be congratulated on having contributed a work of great value to the literature of obstetrics.

[1] "A Manual of Midwifery for Students and Practitioners," by Henry Jellett, B.A., M.D. (Dublin), F.R.C.P.I., L.M., and David G. Madill, B.A., M.B., B.Ch., B.A.O. (Dublin), L.M.; Fourth Edition; 1929. London: Baillière, Tindall and Cox. Royal 8vo., pp. 1293, with illustrations. Price: 25s. net.

THE VEGETATIVE NERVOUS SYSTEM AND CLINICAL MEDICINE.

DR. POTTENGER'S "Symptoms of Visceral Disease" evidently has met with a favourable reception, as it has entered on a fourth edition since its first appearance in 1919.[1]

The theme of the book is the importance of the vegetative nervous system as a mediary in the production of the local symptoms and the general manifestations of disease. The author has drawn on a wide field of literature in marshalling her facts and a bibliography is appended to each chapter. She demonstrates successfully that an appreciative understanding of the sensory, motor and trophic "reflexes" not only aids intelligent interpretation of symptoms, but also stimulates observation. The syndromes of various emotional and toxic states also are reviewed as the expressions of sympathetic or parasympathetic dominance. The more speculative association of calcium and hydrogen ion excess with sympathetic stimulation and of potassium and hydroxyl ion excess with parasympathetic stimulation is stated to have been proved.

The anatomy, physiology and detailed consideration of the vegetative reflexes give the book a considerable clinical value. There is, however, an over-emphasis of the distinction that the sympathetic reflex is manifested mainly in somatic effects, the parasympathetic chiefly in visceral effects. This attitude probably accounts for the relatively slight consideration given to parasympathetic-somatic reflexes and may be responsible for an oversight, the tabulation of the hypoglossal as a parasympathetic nerve. A statement that a vegetative reflex is slow, a somatic reflex rapid, is not entirely accurate. The new terms "hypervegetonia" and "craniobulbar" are not commendable.

When the author leaves her *terra cognita* of the vegetative system, she is often hopelessly at sea. In touching on psychology she expresses the relationship of mind and body in terms of crude interactionism. Perception is classed as an emotion. "Psychic state" is held responsible for the physiological effects of emotion and then it is implied that the lower animals do not experience these to an important degree. Elsewhere these effects in animals are described fully. In discussing the somatic neuro-muscular system the author states that all action and inhibition of action in this system are performed through the will. The nerve path for the reflex withdrawal of the foot from a harmful stimulus is said to pass by way of the sensory and motor cortex. The knee jerk is described as a spinal reflex. It is asserted that the simple somatic reflexes are not found commonly in clinical medicine.

[1] "Symptoms of Visceral Disease: A Study of the Vegetative Nervous System in its Relationship to Clinical Medicine," by Francis Marion Pottenger, A.M., M.D., LL.D., F.A.C.P.; Fourth Edition; 1930. St. Louis: The C. V. Mosby Company; Melbourne: Stirling and Company. Royal 8vo., pp. 426, with eighty-seven text illustrations and ten colour plates. Price: $7.50 net.

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Disseminated Sclerosis.

IN spite of the volume of work which has been carried out on the causation of disseminated sclerosis, the manner in which the typical pathological changes are produced, and the nature of the factor giving rise to them have not been determined. The view most generally accepted is that the lesions are the result of an infective process and unsuccessful efforts have been made to incriminate various agents. The last occasion on which the subject was discussed in this journal was in June, 1924, when reference was made to an important communication by Symmonds. Symmonds made a histopathological study and though he came to definite conclusions, it could not be expected that his methods would yield definite information. The subject has been attacked at Westminster Hospital, London, by Miss Kathleen Chevassut in what promises to be one of the most important bacteriological investigations of recent years. Her findings have been recorded, together with those of other workers, in *The Lancet* of March 15 and 22, 1930.

Miss Chevassut was led to carry out cultural experiments with cerebro-spinal fluid as a result of certain observations on the response to the gold sol test in cases of disseminated sclerosis. She found that 77% of 189 patients with disseminated sclerosis had cerebro-spinal fluid which gave a specific curve, and argued that it was most likely that the gold curve was related in some way to the causal factor, toxic or otherwise. After trying numerous media, she had success with Hartley's broth to which human serum had been added. From the ordinary bacteriological point of view attempts at culture were fruitless, but there was evidence of some change which had not been obtained under other conditions. A change in reaction occurred and this change in reaction was specific. It occurred only in tubes containing cerebro-spinal fluid from patients with disseminated sclerosis and did not occur in control tubes inoculated with cerebro-spinal fluid from persons with conditions other than disseminated sclerosis or in tubes containing uninoculated serum and medium. By a special method of microscopical examination with a dark-ground illumination Miss Chevassut discovered small groups or colonies of spherical bodies some of which appeared to have small refractile granules attached to them. These granules passed through a collodion membrane and when the filtrate was inoculated into serum broth and incubated, spheres and granules made their appearance. Miss Chevassut was able to grow this virus from the cerebro-spinal fluid of 176 patients with disseminated sclerosis from among a total of 188 examined. It was not grown once from 269 control specimens of cerebro-spinal fluid. The persons in the control group consisted of normal persons and those suffering from such conditions as hysteria, *tabes dorsalis*, cerebro-spinal syphilis, subacute combined degeneration of the cord, transverse myelitis, spinal compression, epilepsy, cerebral tumour and encephalitis.

The question which naturally arises, is whether this virus is the cause of disseminated sclerosis. It must be pointed out that Miss Chevassut regards her communication as a preliminary one and that she wisely refrains from making dogmatic deductions. It must also be recognized that the amount of work carried out by her has been colossal. She quotes five experimental observations in support of her contention that the appearance is due to a living virus: (i) Multiplication *in vitro* occurs; (ii) subculturing can be effected; (iii) sensitivity to hydrogen ion concentration, temperature and certain chemical agents is manifest; (iv) sugar reactions, at least in their initial phases, are induced by actively growing cultures, although subsequently they may be due to some other factor; (v) change in the hydrogen ion concentration of the medium results from and is associated with growth of the culture. Koch's postulates have not been satisfied. According to the first of these the microorganism must be present in all cases of the disease. The percentage of cases in which the spherical bodies were recovered, is very high and further when a remission of the disease was brought about (or

occurred) no virus was recovered from the cerebrospinal fluid. According to the second of Koch's postulates the microorganism must be cultivated in pure culture. This has been done in this instance. The third postulate states that the inoculation of the microorganism must produce the disease in susceptible animals and the fourth is that it must be obtained again from such animals and be cultivated in pure culture. Among this series of papers is one by Dr. J. A. Braxton Hicks, Dr. F. D. M. Hocking and Sir James Purves-Stewart. They inoculated the virus into a small series of seven monkeys. In one monkey there developed paralytic symptoms and in this animal the cord manifested well marked Marchi degeneration in one posterior column and in one direct cerebellar tract. In another monkey there was degeneration in one antero-median column, together with a few scattered fibres of degeneration in both lateral columns. These conditions were, of course, not regarded as constituting disseminated sclerosis. It should be stated also that Sir James Purves-Stewart also reports satisfactory results of the use of vaccines prepared from this virus (*Spherula insularis*); they need not be further discussed, for they have no direct bearing on the question under consideration and the author himself advises great caution in their acceptance as definitely valuable.

There can be no doubt that Miss Chevassut has carried out a piece of work of extraordinary value. Other workers will certainly endeavour to confirm her findings. Confirmation will not settle the question of the causative relationship of the *Spherula insularis*. Since her work is only a preliminary report, she will doubtless continue her researches. Extension of her work may yield conclusive results. Another aspect not to be forgotten is that this research opens up avenues for increasing our knowledge of the filtrable viruses.

Current Comment.

ANGINA PECTORIS.

THERE are many and varied views as to the pathogenesis of *angina pectoris*. Clifford Allbutt considered that the phenomena were generally due to tension of the first part of the aorta when it is the seat of some inflammatory or degenerative lesion. The sudden death that so frequently occurs, is due in his opinion to reflex vagus inhibition. James Mackenzie, on the other hand, held that *angina pectoris* is "an expression of cardiac exhaustion and also an expression of a susceptible nervous system" and that the pain is a viscera-sensory reflex phenomenon. Probably the most generally accepted view is that *angina pectoris* is due to anoxæmia of the heart muscle which results from the inability of diseased coronary vessels to deal with increased demand, from coronary spasm or from a combination of both. Thomas McCrae discusses *angina pectoris* after a careful study of a number of patients.[1] He first of all emphasizes the importance of separating acute coronary occlusion (cardiac infarct) with its definite clinical features and positive lesion, from *angina pectoris* with its vague pathogenesis. He then sets out to confound the prevalent idea that *angina pectoris* is always due to coronary involvement. He tells of several patients who had repeated attacks of angina referred to the upper part of the sternum and radiating into one or both arms, followed by a typical attack of acute coronary artery occlusion. The pain of the latter was in the lower part of the sternum, did not radiate and was said by the patients to be quite different in character from the former pains. In a few patients with definite aortic lesions, attacks of anginal pain occurred which were situated in the upper part of the sternum and radiated. McCrae believes that in these patients the upper sternal pain was of aortic origin. He then tells of a patient who had anginal attacks for fourteen years, the pulse rate remaining between seventy and eighty during attacks and the blood pressure scarcely altering from a systolic figure of 140 and a diastolic of 90 milligrammes of mercury and the heart showing no clinical evidence of abnormality. The patient died in an attack. McCrae submits that if all these attacks had been due to coronary artery disease, the myocardium would surely have been damaged to a considerable extent and that if they were due to coronary spasm, the heart's action would have been more affected. Other cases are described in which the anginal attacks were associated with œsophageal or gastric symptoms. In one patient the passage of a stomach tube invariably relieved the attack. In other patients the removal of gall stones resulted in the cessation of attacks. The author admits the possibility of coronary spasm in these patients, but believes rather that a purely nervous mechanism was at work. He concludes that the evidence does not support the statement that *angina pectoris* is always due to coronary artery disease; that there are probably several factors which may be operative in causing an attack in the aorta, in the coronary arteries and myocardium and in the nervous system; that it is doubtful if disease of any one structure can explain all cases.

[1] *American Journal of the Medical Sciences*, January, 1930.

Disease of the aorta and coronary artery disease or narrowing are so frequently associated that it is impossible to divide them arbitrarily as causative factors and it is also impossible to determine on clinical evidence alone to what extent the coronary arteries and myocardium may be involved. Here is where McCrae has failed to gain his point, for he gives no *post mortem* evidence to support his contention that in many of his patients the coronary arteries and myocardium were not affected. With regard to the patients whose attacks were relieved by the passage of a stomach tube or the removal of gall stones, they were not suffering from true angina, but from what is known as pseudo-angina. The pain in these circumstances is to be regarded as a viscero-sensory reflex phenomenon. It is interesting to note the recent experiments of S. A. Levine, C. Ernstene and B. M. Jacobson on injections of adrenalin as a diagnostic test for *angina pectoris*.[1] These investigators found that an injection of one cubic centimetre of adrenalin produced a typical attack in ten out of eleven patients who were sufferers from *angina pectoris*. Though they admit that the pathogenesis of angina is still obscure, they favour the view that it is due to anoxæmia of a restricted part of the myocardium. The injection of adrenalin increases the work of the heart owing to a consequent rise in blood pressure and an increased metabolism. It also increases the coronary blood flow. In normal persons the increased oxygen requirement of the myocardium is probably compensated for by the increased coronary flow. When, however, the coronary circulation before injection is below normal, then full compensation cannot take place. The production of a localized myocardial anoxæmia therefore is easily understood after the injection of adrenalin into patients whose coronary circulation is impaired by disease of the aorta or coronary arteries. This is in favour of the coronary theory as to the causation of *angina pectoris*. It should also serve to differentiate true from pseudo-angina.

[1] *Archives of Internal Medicine*, February, 1930.

PRIMARY MALIGNANT INTRATHORACIC TUMOURS.

THERE can be no doubt that increasing attention is being paid to the study of primary malignant intrathoracic tumours. This is possibly due to the occasional successes which have attended the use of deep X ray therapy and to the modern development of surgery of the thorax. It is often stated in the literature that malignant tumours of the thorax are occurring with greater frequency. This impression may be due to the more intensive study which is being given to them. These tumours are of interest from both the clinical and the pathological aspects. That tumours of the lung and mediastinum can produce a clinical picture of extraordinary interest and complexity was shown in a report by Ingram and Walton Smith in this journal on January 4, 1930. The tumour in this instance was thought to be an embryoma and the specimen presented a unique appearance. An interesting review of 139 cases of carcinoma of the lung was published in April, 1929, by S. L. Simpson in *The Quarterly Journal of Medicine*. This article is worthy of careful study. Another series of 239 primary malignant intrathoracic tumours has been studied by James Maxwell.[1] Of these tumours 184 were classified as primary bronchial carcinomata, 37 were classified as arising primarily within the mediastinum and five were looked on as primary pleural growths. Thirteen tumours could not be classified at all. The greatest interest attaches to the 184 primary bronchial carcinomata. Fifteen of this number are described as consisting of a local tumour surrounding a main bronchus without obvious infiltration of either lung or mediastinum. In 49 instances the tumour had arisen in the bronchus and had proceeded directly to infiltrate the corresponding portion of the lung. In this type the infiltrative process was of two varieties. Either the malignant cells spread directly along the course of the peribronchial lymphatics encircling the bronchi in a continuous sheath of neoplastic tissue, or the process extended further and the lung tissue was infiltrated and destroyed, and the malignant tissue could be seen replacing the alveoli themselves. In thirty-six instances the tumour had arisen from a main bronchus and instead of infiltrating the lung, had extended directly outwards into the mediastinal tissue. It is obvious that tumours of this variety might easily be regarded as originating in the mediastinum. Maxwell, however, holds that when a main bronchus is grossly involved in malignant growth, the tumour is in the nature of a bronchial carcinoma. He gives good reasons for this view and asks the pertinent question as to why, if the growth really arises in the mediastinum, the infiltration should be as a rule unilateral. In eighty-four instances (the largest group) the tumour had started in the main bronchus and had spread into the surrounding lung tissue and also into the mediastinum. The microscopical appearances of one hundred and eleven of these tumours are discussed. Of these forty-seven had the characteristics of obvious carcinomata and sixty-four had those of a small oval-celled tumour—the so-called oat-celled tumour. In regard to the latter type of tumour there has been much discussion. For many years they were regarded as sarcomatous. In 1926 Barnard produced evidence pointing to their carcinomatous nature and Simpson, in the paper previously mentioned, came to the same conclusion. Maxwell admits the carcinomatous nature of these tumours, but does not believe that they arise from exactly the same cells as the obvious carcinomata; he gives good reasons for the view that they arise from the basal layer of the bronchial epithelium. It is interesting to note that Maxwell found no evidence that any of the tumours in his series arose directly from the epithelial lining of the pulmonary alveoli; Ewing states that pulmonary carcinomata may arise from alveolar epithelium.

[1] *The Journal of Pathology and Bacteriology*, April, 1930.

Abstracts from Current Medical Literature.

SURGERY.

Thoracic and Lumbar Sympathetic Ganglionectomy in Peripheral Vascular Diseases.

ALFRED W. ADSON AND GEORGE E. BROWN (*The Journal of the American Medical Association*, January 25, 1930) in a paper on thoracic and lumbar sympathetic ganglionectomy in peripheral vascular diseases come to the following conclusions. Sympathetic ganglionectomy and trunk resection constitute a surgical procedure of considerable magnitude which surgeons are justified in using in the treatment of advanced Raynaud's disease, in the early developing vasospastic cases of scleroderma and in *thromboangiitis obliterans* in which vasospasm of the collateral arteries exists. The operation is probably indicated in allied and border-line cases, but should be employed with caution, for the procedure is not a cure-all for all peripheral vascular diseases.

The Operative Treatment of Pylorospasm in Infants.

H. VON HABERER (*Deutsche Medizinische Wochenschrift*, December 6, 1929) discusses the operative treatment of pylorospasm in infants. He has adopted some important modifications in the operative technique recommended by Kirschner. He advocates a small mid-line incision immediately below the xiphoid cartilage of the sternum. This exposes the liver which is retracted upwards, and the large hypertrophied pylorus and antral portions of the stomach come into view. The whole of the contracted portion is seized between the thumb and forefinger of the left hand, so that the forefinger invaginates the anterior wall of the stomach into the pylorus and the thumb pushes the anterior wall of the duodenum up against it. With a very sharp knife he then divides all the muscle fibres completely down to the mucous membrane, so that the mucosa bulges out through the gap thus made in the muscular layer. Great care must be taken not to injure the mucous membrane, especially on the duodenal side of the pylorus where the muscular layer is thin. In order to relieve the spasm completely, the muscle fibres must be divided well into the antrum of the stomach. At the time of division these fibres appear white and bloodless and no hæmorrhage is seen from the incision, but when the spasm has been released, reactionary bleeding may occur, as happened in the author's second case after the stomach had been replaced. Hence he advocates pausing for a few minutes to allow time for any hæmorrhage to manifest itself and be dealt with by ligaturing or even sewing together the serous layers of the incision before the viscus is replaced in the abdomen. When this has been done, the liver falls back into position to cover over the stomach and the wound can be closed rapidly. All his patients had been subjected to intensive conservative treatment by pædiatricians beforehand for some time; many indeed were in advanced stages of the condition, yet after an average convalescence of four weeks they were ready to be handed over in excellent condition to their parents. Immediately after operation the infant is given over to the pædiatrician who has complete charge of all after-treatment, feeding *et cetera*, except of the wound and any surgical complications which may occur. Thus his technique is as simple as possible; he sutures the wound with fine catgut, covers it with silver foil to seal it up and fixes the dressing on securely with sticking plaster. As a result no wound infections have occurred nor any prolapse due to breaking down of the wound. He stresses the importance of making the incision as described above, in order that the liver may come back into place between the stomach and the abdominal wall, to prevent secondary hernia in the wound which might easily occur in such atrophic under-nourished children. The pædiatricians commence feeding with the mother's milk two hours after the operation; if it is tolerated, the amount is rapidly increased. Saline solution is given *per rectum* by the drip method for twenty-four hours. Usually the result of the operation is immediately evident and no further vomiting occurs. When it does occur, it is usually due to the operation not having been done properly and some muscle fibres having been left undivided. The author has had only one patient with this type of post-operative vomiting and this had readily stopped when the quantity given at each feeding was reduced slightly; the total daily number of feedings remained the same. Daily weight and feeding charts are advocated as being most important and should be inspected every day. Very occasionally a patient is met with who vomits, mostly old blood, during the first day after the operation, however carefully the nourishment is given. A single gastric lavage is all that is necessary to stop this. It was found to occur in infants whose stomachs had shown extreme hyperperistalsis prior to operation, but which had disappeared at the time of operation and left behind a condition of atony of the stomach musculature with numerous small erosions in the mucous membrane.

The Problem of Pylorospasm.

WILHELM BAYER (*Deutsche Medizinische Wochenschrift*, December 6, 1929), despite the present day conviction that operation is the treatment of choice for pylorospasm in infants, quotes the result in eighty-six patients treated at the University Clinic, Berlin, to show that among the forty patients treated by medical measures only one death occurred. Among the forty-six patients treated by the Weber-Ramstedt operation twelve deaths occurred, mostly soon after operation, from pulmonary infections. He points out that the indications for operation must be considered with great care and that the condition of the child at the time of operation affords a better guide to prognosis than the skill of the surgeon. When the loss of weight since birth has been rapid and extensive, operation is most dangerous. Of eighty-six patients seventy-eight were males and yet recent researches into the problem of the causation of pylorospasm have proved that the influence of the maternal ovarian and anterior lobe pituitary hormones which are found in the blood of the umbilical cord, is not a determining factor, because their excretion in the urine of both male and female infants is complete in five days. He draws attention to the fact that pylorospasm follows a seasonal curve, 75% of the cases occurring between the months of January and June. This closely resembles the seasonal curve of another malady of infancy, namely, spasmophilia, and thus parathyreoid glandular therapy may be worth a trial. He concludes with a reference to the vitamin theory and Reyher's claims of cures with vitamin *B*.

Treatment of Acute Osteomyelitis.

D. CANON (*Deutsche Medizinische Wochenschrift*, February 7, 1930) considers that, if possible, all patients with severe acute osteomyelitis should be given serum from convalescents—a course naturally only possible in big clinics. Failing this, an autogenous vaccine should be prepared at once and its use will materially assist recovery. A course of such a vaccine is recommended before any radical bone operation undertaken after the acute symptoms have subsided. In subacute periostitis and osteomyelitis favourable results follow vaccine therapy in association with exposure to the sun or quartz lamp.

Pyloric Stenosis.

B. ALBRECHT (*Deutsche Medizinische Wochenschrift*, January 17, 1930) discusses the anatomical changes seen in cases of pyloric stenosis and then describes a modification of the Ramstedt operation which he has found to be of considerable value. He makes four longitudinal incisions in the region of the pylorus and the first part of the duodenum. These incisions pass through the serous coat well into the muscular layer. By this method the lumen is better dilated than with one incision. Œdema quickly disappears and the infant can retain food by the day following operation.

The Thyreoid Lymph System.

G. S. WILLIAMSON AND I. H. PEARSE (*The British Journal of Surgery*, January, 1930) write about the comparative and embryological anatomy of the special thyreoid lymph system, showing its relations to the thymus, with some physiological and clinical considerations that follow therefrom. A special thyreo-thymic lymph system

is described as the essential and most primitive feature of thyreoid anatomy. The intrathyreoidal portion of this system consists of thyreoid lymph sinusoids draining into intralobular lymphatics. These lymphatics, unlike those of the capsule of the thyreoid, emerge from the substance of the gland at the hilum of each lobe. The extrathyreoidal portion of this system consists of lymphatic channels proceeding from the hilum of the thyreoid lobe to their termination in thymic tissue. Thymic tissue occupies both neck and mediastinum, thymic nodes being liable to occur anywhere witnin a special deep fascial compartment of the neck extending from the base of the skull to the pericardium. Structurally all thymic tissue is formed of the coiled plicated capillary terminals of the thyreo-thymic lymphatics. The thymus is thus essentially of the nature of a lymph reservoir to the thyreoid gland. A special secretion of the thyreoid which has been called lymphogenic secretion, is poured into the thyreoid lymph sinusoids and can under appropriate conditions be seen pervading the whole thyreo-thymic lymph system. In the primitive form the thyreoid lymph reservoir drains by a valved opening directly into the heart. The thyreoid and thymus must be regarded as one apparatus in all considerations of the thyreoid function. Especially must this apply to the study of Graves's disease, thyreotoxicosis and endemic goitre. The lymphogenic secretion of the thyreoid (which elsewhere we have shown to be responsible for thyreotoxicosis) is normally detoxicated in the thyreothymic lymph channels. This explains the constant association of Graves's disease and thyreotoxicosis with *status thymico-lymphaticus*. It also explains the claims made for thymectomy in the treatment of thyreotoxicosis. The thyreo-thymic lymph system is a closed system. It has no connexion with the cervical lymphatic glands into which the *lymphæ comites* of the capsular veins of the thyreoid drain. Thyreoid cancer occupying the thyreo-thymic lymph channels may appear in the neck or mediastinum, since this is the area over which thymic tissue is normally distributed. Under such circumstances the growth is still confined within the organ of its origin and is still capable of function. Such intrathymic nodules are not to be classified, *qua* malignancy, in the same category as the "cellular seeds" of true metastases found in the ordinary lymph glands. Certain features of the cystic hygromas of the neck suggest that these tumours have origin in developmental abnormalities of the thyreo-thymic lymph system.

Spontaneous Rupture of the Normal Spleen.

HAMILTON BAILEY (*The British Journal of Surgery*, January, 1930) writes about the spontaneous rupture of the normal spleen. There are many who doubt that a normal spleen can rupture spontaneously. There are but eleven recorded cases of the condition. There are at least three examples in which after successful splenectomy for supposedly spontaneous rupture the investigator has been able to elicit the history of a blow. No such history was obtainable in his case. An accident of sufficient magnitude to rupture a normal spleen is unlikely to be forgotten by the recipient. There is a good aphorism: "In a traumatic hæmoperitoneum in the male examine first the spleen." On the clinical side spontaneous rupture of the splenic vein bears comparison with spontaneous rupture of the spleen; pathologically the conditions are widely separated. Kehr's sign of left shoulder pain in ruptured spleen was well marked, although its diagnostic significance was not appreciated. Yet another example of hæmoperitoneum with a comparatively slow pulse rate is brought to notice. Additional evidence is afforded that the mid-line upper abdominal incision is adequate for splenectomy for rupture of the spleen.

Periarterial Sympathectomy.

L. ROGERS AND A. HEMINGWAY (*The British Journal of Surgery*, January, 1930) give their results of an experimental investigation of the effects of periarterial sympathectomy. Although Leriche and his pupils have done much experimental work, at the present time periarterial sympathectomy may be described as an empirical operation, since its performance is based almost entirely on the favourable results observed clinically in certain cases. An experimental investigation has been carried out of the effects of periarterial sympathectomy in animals. A description of the methods employed for the destruction of the periarterial nerve network is given. The effect of periarterial sympathectomy performed upon the femoral or femoral and popliteal arteries of the cat, has been measured by comparison of the responses to certain substances of the vessels of the limb operated upon with those of the corresponding normal limb. Vasodilatation follows the operation, but is very transient. Comparison of the heat production in the limbs of the cat following the performance of the operation on one side, indicates that no permanent vasodilatation results. Periarterial sympathectomy performed upon the carotid artery of the albino rabbit results in a vasodilatation of the corresponding ear lasting for about forty-eight hours and then disappearing. Division of the main sympathetic trunk produces a greater and more lasting vasodilatation than periarterial sympathectomy of the corresponding main artery.

Alcohol Injection of Hæmorrhoids.

H. ELSNER (*Deutsche Medizinische Wochenschrift*, January 31, 1930) describes his results in a series of one hundred cases of hæmorrhoids treated with injections of alcohol. As these injections are very painful a solution of "Novocain" and adrenalin is used first and the alcohol is injected ten minutes later. On an average 0·5 to 1·0 cubic centimetre is sufficient for each mass and the mass should be returned to the anal canal after injection. While the treatment may be ambulatory, it is better to keep the patient in bed for three to four days. Castor oil is given on the third night. There may be slight hæmorrhage for one or two days. Naturally the bowels are kept freely open. Warm sitz baths will overcome any tenesmus in the first few days. The author has seen no complications with this method of treatment and the treatment is particularly advantageous in patients with general disease precluding surgical procedures.

Surgical Treatment of Angina Pectoris.

W. DENK (*Wiener Medizinische Wochenschrift*, January 1, 1930) considers that the indications for surgical interference in *angina pectoris* are not yet clearly defined. The total reported cases up to 1927 were 135 with success in 65%, failure in 17% and a mortality of 13%. He prefers the Singer operation, whereby the posterior nerve roots of the lower cervical and the upper four dorsal nerves are divided. The history of a case is given in detail. The severe daily anginal attacks noted before operation completely disappeared and the patient was able to return to work. He has now been twenty months under observation without recurrence. Gastric crises which were present, have naturally not been affected; possibly if the operation had been extended as far as the eighth dorsal roots, this would have been abolished. The operation is a severe one and not suitable for patients with pronounced myocardial degeneration. For this type of case he recommends cervical sympathectomy.

Multiple Polyposis of the Colon.

J. H. ANDERSON AND O. A. MARXER (*The British Journal of Surgery*, January, 1930) writes about two cases of multiple polyposis of the colon in which the diagnosis was made by X rays as well as by sigmoidoscopy. The radiological appearances and technique are described and discussed. In one instance the accompanying colitis was acute, in the other it was chronic with exacerbations. Other members of the family of the first patient had suffered from ulcerative colitis, cancer of the rectum and cancer of the cervix. This supports the experiences of D. H. Pennant and J. P. Lockhart-Mummery. The general colitic nature given by the first patient, especially the stools, is contrasted with the clinical picture given by the second patient in which it is surmised that the polypi were of longer standing, and they were thought to be true tumours rather than merely inflammatory in origin. Whilst the anæmia was greater in the second patient, the nutrition was better. It would appear that polyposis gives rise to less abdominal pain and interferes less with nutrition than colitis, but is more liable to cause bleeding. In each instance constipation was an early symptom, if not the earliest.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE EAR, NOSE AND THROAT SECTION OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Sydney Hospital on April 11, 1930, DR. GARNET HALLORAN in the chair.

Epithelioma of the Larynx.

DR. DOUGLAS CARRUTHERS showed a patient with epithelioma of the larynx involving the anterior commissure and the anterior third of both vocal cords. The history was of hoarseness for six weeks; there were no other symptoms. The growth did not appear to involve the subglottic region, but there was some suggestion of involvement of the stem of the epiglottis in the deeper unulcerated portion of the growth; no glands were palpable. Wassermann and tuberculosis tests gave no reaction; the lungs were apparently sound. Dr. Carruthers proposed to do window resection of the thyreoid alæ and insert a palisade of radium needles as practised by Harmer. This he considered advisable for at least a short trial rather than immediate laryngectomy, total or partial. However, since the condition was bilateral and radium would have to be placed on both sides, serious consideration had to be given to the greater probability of cartilage necrosis in the remaining framework of the thyreoid owing to cross-fire action.

DR. N. H. MEACLE supported the method proposed, pointing out that it was essential, if necrosis be avoided, to use needles free of radium at either end, since the ends of the needles were to be inserted under the remaining cartilage margins.

DR. J. P. FINDLAY and DR. A. L. CLOWES both supported Harmer's method of window resection followed by radium which might be applied in a dental compound collar to give radium effect at a distance.

DR. J. WOODBURN had not seen the patient, but expressed the opinion that if, as Dr. Carruthers suspected, the growth involved the epiglottic stem, total laryngectomy was the only thing to do, this to be followed by radium. He further pointed out that tracheotomy was not necessary in window resections nor with resection of only one cord by thyreo-fissure; instruments for emergency opening of the trachea, of course, should always be at hand. It was thought that tracheotomy, especially if too high, was liable to contribute to after stenosis.

DR. GARNET HALLORAN considered the growth intrinsic and advised Harmer's window resection followed by radium; then after waiting three months total laryngectomy might be considered.

Nasal Cyst.

Dr. Carruthers's second patient was a woman who complained of a swelling in the region of the right naso-labial fold and right nasal obstruction of varying degree for six years. The swelling varied in size from time to time and lately had been increasing; there was no pain and no epistaxis. On examination a bluish, translucent cyst was seen on the floor and outer wall of the anterior choana, its covering membrane being continuous with the mucosa of the floor of the nose and the skin of the vestibule of the nostril. The cyst could be felt high up under the lip and appeared to lie in a recess of the maxilla at the choanal margin. X ray examination had suggested a polypus in the right antrum in a position corresponding to the cyst. The diagnosis lay between dentigerous cyst, mucocele or fibroma. Dr. Carruthers proposed to attempt removal through an incision under the lip, if necessary entering the antrum through the choanal margin as in Denker's method.

DR. A. L. CLOWES considered the cyst probably of dental origin.

DR. J. WOODBURN had seen a somewhat similar case in which the cyst had been incised and curetted. When last seen the condition was settling down, but the patient had not reported for a final inspection. He considered the condition in his patient to have been a mucocele.

DR. GARNET HALLORAN referred to swellings which he had seen, exhibiting the possible great extension of dentigerous cysts and their site in locations in the maxilla which might be misleading, so that dental origin might not be suspected; in one patient on whom he had operated, the cyst extended to the orbital floor, yet it contained rudimentary teeth.

Aneurysm of the Lingual Artery.

DR. GARNET HALLORAN presented a patient who had suffered from a profuse hæmorrhage from the mouth. Examination revealed a soft round tumour, involving the right side of the tongue in line with the fauces. Examination under anæsthesia revealed a distinct thrill in the tumour and blood had been withdrawn from it with a needle; the diagnosis was aneurysm, apparently of the right lingual artery. Dr. Halloran pointed out the necessity for much consideration before deciding upon a method of operation to effect a cure; opinions had been expressed that nothing short of ligature of the external carotid artery and approach to the tongue by resection of the angle of the mandible would be advisable in this case.

DR. D. CARRUTHERS advised exposure and ligature of the lingual artery by an external route.

DR. J. WOODBURN pointed out as instance of accessibility, where necessary, that he had seen the lingual artery injured in the mouth and it had been picked up and tied at the site of injury. In Dr. Halloran's case the aneurysm itself lay in the way of such an approach.

Cancer of the Tonsil.

Dr. Halloran's second patient had been referred to him by and was shown by courtesy of Dr. Sandes. The patient had been sent to him in the Ear, Nose and Throat Department for radium insertions into a cancer which had originated in the tonsil and now extended into the tongue. He was shown on this occasion to demonstrate the radium reaction after nine days. Dr. Halloran stressed the necessity for skilled examination in detail of throat and pharyngeal cancers. In this case his examination had shown the growth to extend into the glosso-epiglottic fossa and, in fact, it was seen to extend across the midline of the posterior surface of the tongue, an important factor in deciding the extent of any radical operation for its removal; it was also stressed that in order to secure proper access for radium implantation in such cases suspension methods were necessary.

Naso-Pharyngeal Fibroma.

DR. J. WOODBURN exhibited a specimen of naso-pharyngeal fibroma removed from an elderly patient. The growth had been approached by palatal fissure and had broken off very easily from its base. Later examination had revealed malignant changes in the base and radium had been applied and held in place by packing. Dr. Woodburn discussed the origin of such growths, pointing out that this appeared to have its origin in the posterior ethmoid cells; these, with the sphenoids and antrum, had been easily reached by the palatal incision.

General discussion as to the site of such growths followed, it being agreed that they might arise from the margins of the choanal and posterior sinuses, as well as from the periosteum of the basi-sphenoid, as was commonly described.

Frontal Sinusitis and Brain Abscess.

DR. D. CARRUTHERS demonstrated a patient for Dr. H. Seaward Marsh. The patient was a boy who had had an acute frontal sinusitis, at first believed by those at home to be due to an insect bite. At operation there had been much pus in a large sinus. A second operation, done on account of persistent discharge, had revealed a perforation in the posterior wall and on enlarging this a large collection of pus had been found within the *dura mater* and presumably in the brain itself. As seen now, the wound was dry and almost healed and the patient manifested no ill effects. In order to secure wide exposure of the

sinus which extended to the opposite side, Dr. Marsh had preferred to carry the usual frontal sinus incision across the nasal bridge and into the supraorbital ridge of the other side, rather than adopt the formerly practised method of extending the incision vertically from its nasal end. The result was a minimum of scarring and very little deformity, while excellent access was obtained and the whole of the anterior wall of the sinus had been removed up to its junction with the posterior wall.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Walker, Norman Arthur, M.B., B.S., 1929 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

The undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Stephens, William Lodewyk Bosschart, M.B., B.S., 1929 (Univ. Melbourne), Alfred Hospital, Prahran, S.1.

Public Health.

VICTORIAN COUNCIL FOR MENTAL HYGIENE.

A MEETING convened to form a Victorian Council for Mental Hygiene was held at the Medical Society's Hall, East Melbourne, on May 2, 1930, SIR RICHARD STAWELL in the chair.

DR. S. V. SEWELL said that the Committee of the Neurology and Psychiatry Section of the Victorian Branch of the British Medical Association did not wish to apologize in any way for calling the meeting nor for taking the initial steps to call it. It had been recognized by practising neurologists and psychiatrists, especially since the war, that a tremendous amount of the nervous disturbances which patients who consulted them were suffering from, was due to the fact that the patients were misfits and that conditions with which they were constantly confronted, were the result of the fact that they did not select their work in life satisfactorily. They all knew perfectly well that the vast majority of children were simply put into some position because the position chanced to be open at the moment. The results of such misfits were seen to an extraordinary extent during the war and led throughout the world to a stirring up of neurologists and psychologists and physicians generally regarding preventive medicine in that particular realm of work. Preventive medicine had long come into its own where bodily ailments were concerned, but had been a very long time in doing so in the most important field of its future work—preventive medicine with regard to the possibility of the later development of psychopathic tendencies in children who would otherwise be comparatively normal.

As a result of that, the Council for Mental Hygiene had been formed in many countries and was doing magnificent work, so that when the suggestion came that such a council should be formed in that community it was welcomed, although it was realized that it could only be successful if there were adequate cooperation between the medical profession, the teaching profession and all social workers. With that end in view the meeting had been called and invitations sent out to many people, although, unfortunately, there were numerous other people overlooked who should have been invited, as was inevitable when things were done in such a hurry.

SIR RICHARD STAWELL said that in order that they might have some clear and helpful idea of the subject, several speakers had been invited by the Organizing Committee to address the meeting upon the aims and scope of work of the Council for Mental Hygiene and to tell them of the work that was being actually done by such councils in other parts of the world, particularly in England. The first speaker to address them would be Professor W. S. Dawson, Professor of Psychiatry in the University of Sydney.

PROFESSOR W. S. DAWSON said that if one considered the history of mental disorders, the ways they had been treated and the general attitude which had been adopted to them, both by the medical profession and by the general public, one realized that it was not so very long ago, only two or three generations, when the whole subject of mental disorder was limited to the treatment of those unfortunate individuals whose disease had progressed to such an extent that they had to be placed in mental hospitals, even against their will, and kept there and treated as best could be until such time as some of them were able to rejoin their fellows in the community and again play their part in society. Interest in and attention to mental disorders were very largely centralized to what happened behind the walls of mental hospitals and there was very little organized attempt to do much for individuals before they had reached the stage at which they had to be certified and treated against their will. Gradually a wider conception of mental disorder had grown up and it was realized that what was termed the severer type of insanity, in which the patient became incapable of realizing there was very much the matter with him, a stage at which he not infrequently became antagonistic towards any attempt to help him, was only a small fraction of the mental maladjustment which occurred in the general body of the community. At the present time they had a very much wider conception of mental trouble, including all forms of maladjustment, cases of mental backwardness and all those forms of mental inadequacy which rendered an individual unfit to play his full part in the general duties and responsibilities of the community.

This, continued Professor Dawson, was the age of prevention and he need hardly remind his hearers of the immense amount of work and achievement that had occurred in the sphere of ordinary public health and general physical hygiene, but with this wider conception of mental trouble, it naturally occurred to many people that patients were handled and seen by specialists far too late in their mental disorders. Nor need he remind them of what might be done if an attempt were made to handle them at a very early stage before severe developments had taken place. Moreover, on tracing back the history of such patients, it was realized that the conditions in which they were brought under medical notice were, in quite a number of instances, merely the culmination of a long series of maladjustments which could sometimes be traced back to the years of adolescence or even childhood. He thought it had been no uncommon experience of many of those who had to deal with such cases, to hear the patient say: "If only I had known that years ago, if only I had understood the reason why I developed this extraordinary antagonism to some other people, the reason why I had a sensitiveness on some point or other, if I could have talked that over and had it explained to me years ago, what a tremendous amount of misery I might have escaped!" Their patients said such things to them occasionally and psychiatrists realized the tremendous importance of tackling these problems in the early years of life; and one of the objects of the preventive side of mental hygiene was very closely associated with this attempt to deal with children who were displaying nervous symptoms. The various fears, emotional upsets and feeling of inferiority and insufficiency in a tremendous number of these patients, if fully dealt with at an early age, could be contended with and would give place to a feeling of self-confidence.

One considered the question of criminality when going over the history of quite a number of these patients. There was a time in the life of many a hardened criminal when he was a decent, law-abiding youth and then through some unfortunate environmental factor or some incident in his life he became turned to the path of antisocial behaviour. That was an aspect into which mental hygiene was attempting to introduce preventive measures and one of the objects which all such organizations had very much before them was a thorough study of juvenile delin-

quents, examined at a stage before they had developed set habits or antisocial tendencies, in order that they might be examined and considered from the environmental aspects. The disturbing factor was not infrequently something in the home, a broken home or some failure of the parents to exercise adequate control.

Childhood had been very aptly termed the golden period for mental hygiene. In dealing with children and disturbances which they might display, the object was to consider these problems from all points of view, not only the physical, but also the mental and psychological questions, their intellectual development and the question how far mental backwardness might contribute to their insufficient responses to their environment. Not only the physical, mental, psychological, but also the environmental factor was one of tremendous importance and they hoped to enlist the services of the social organizations in order that they might have a proper report and supervise such individuals after their examination. The physical aspect had already been very widely recognized—they recognized the enormous importance of a sound body for a sound mind. He might mention such an organization as the Tresillian movement, where the physical health of the child was carefully supervised at a very early stage, and also the method of handling by the parents. That had an enormous influence not only on the physical adequacy of the child, but also on the emotional, as physical ill health would have its repercussions. Then the parental and home influence on the children was of immense importance, as in quite a number of cases of delinquency the children had been insufficiently cared for at home. The various social organizations should deal with that, as every living thing had to react to its environment and in dealing with mental disorders the environmental factor might be of paramount importance. If the Council could not improve or help the individual who had various incapacities in his constitution or mental development, they could certainly do an enormous amount in adjusting the environment and putting the individual into an environment best suited to him.

Industrial hygiene and vocational guidance were of immense importance to the boy who was leaving school after a thorough physical and psychological examination, so that he might be definitely guided as to what type of occupation he was best suited for. The failure of the individual to enter upon the type of career most suitable for him was a potent cause of nervous trouble owing to the maladjustment which was involved. Mental hygiene essentially involved team work; it was not purely a medical question, but was one which should enlist the services and help of not only psychologists and educationalists, but also of all people who were engaged in social welfare work. One of the great difficulties they had to deal with in these mental disorders, was the education of public opinion up to a higher standard as to what should be done by public bodies for the welfare of those who suffered from mental disorders.

Then there was the enormous question of mental deficiency which was a tremendous question in most countries, and one which every country had sooner or later to face and introduce legislation to deal with. Institutions had to be provided where defectives could be cared for and more important was the provision of some sort of training for that group of defectives which could sometimes be turned back into the community after some years of training in special institutions or given special occupation and supervision, particularly over the difficult years of adolescence, so that after attaining a certain amount of stability they could play their part in the community.

Then there was the question of providing for special cases in the educational system. This was a point which had been very strongly emphasized after a survey recently made by a committee in England. Some years before the war there had been a Royal Commission on Mental Deficiency in England which presented statistics that alarmed the community. A much more detailed survey had been made within the last three or four years and their report last year stated that a good deal more should be done by way of provision for such cases, so that children who were two or three years mentally retarded should be given special types of training. It was felt that they should not be sent to special institutions, but after a special educational training they would be of value to the community. He was, continued Professor Dawson, correct, he thought, in saying that Mr. Henry Ford maintained that he could provide employment at some comparatively trifling thing for 10% of imbeciles in his works. It was a great thing to go on, that everybody, even of the most limited capacity, could do some sort of useful work. There had been far too much pessimism in feeling that nothing could be done for defectives. With patience and with special facilities, many of those individuals could become useful in the community. That was a point whereon public opinion must be educated and the legislature encouraged to make special provision for such persons—for Parliament would never do anything unless public opinion was behind it—and was one of the most important objects of mental hygiene organization. Pressure must be brought to bear so that the necessary legal measures might be instituted.

The term "movement" had been rather unfortunately applied to mental hygiene. There was no sense of motion about it. He felt very strongly that medical men could guide the community regarding the problem of mental disorders, but the medical profession must be helped by the social workers, psychologists, educationalists, magistrates, especially those who had to do with children's courts, and the legal profession, in order that they might all get together and consider the problem from every aspect and make their recommendations with due consideration, trying to educate public opinion to have them carried through.

In the United States of America there was a large organization and next week they would have there the first International Conference, at which various countries would be represented, including Australia. In Britain a National Council had been formed in 1922 and in most countries, including the lesser ones, there were mental hygiene organizations which attempted to work more or less on the lines he had indicated, although, of course, there were many local modifications necessary. He regretted to say that in New South Wales they had done nothing very much yet except consider the question, but had had a meeting at which representatives from various social bodies, welfare organizations and societies, representatives of the National Council of Women, of the Children's Court, of the University, of the Education Department and psychologists had all met and decided that the time was ripe for the formation of such an organization. A combined meeting had been held at the Australasian Medical Congress which various representatives from lay bodies attended, when all the speakers emphasized the importance of mental hygiene in the wider treatment of mental disorders. From what he had heard in Melbourne, it seemed that people were very keen and that great events were in view.

PROFESSOR J. A. GUNN, upon being asked by the Chairman to place his views before the meeting, said that after the very interesting and profound, though brief, outline given by Professor Dawson, he did not feel that there was much that he could say, except in the first place to endorse the importance of the meeting and all that it stood for. It recalled to his mind the discussions which he, as a layman, had had in Australia, but more particularly in England and France, with men of his own age who were going through college and who were in the medical profession. He thought of evenings in which they had discussed the problems of the individual and the happiness of the individual, the question of physique and of mental outlook and of fitness for the job. They used to dream that a time would come when there would be closer cooperation between the medical profession, the educationalists and the social workers, each of whom saw these problems from their own particular angle. He thought that the meeting that evening was an historical occasion for the City of Melbourne in that it was highly representative of the different orders and associations. He intended to make only one or two comments of a very brief kind. Professor Dawson had referred to the fact that next week there was to be held the first International Congress in the United States of America to which various countries of the world were sending representatives, medical and non-medical. From his own point of view, which was that of

the psychologist and more particularly that of a student of social affairs, the importance of a meeting of that kind was that it served to direct attention to a problem of their own age which people were apt to miss if they talked in large generalities about progress and legislation and economics, overlooking the test of any civilization —what kind of lives people were living; to put it simply and crudely, were they happy? Happiness implied harmony of the individual with his environment, and mental hygiene was devoting its attention to individuals who, often through no fault of their own, were ill adapted to their environment, very often at the start and later in their schools and their workshops. Anything which could prevent that tremendous disaster which was represented by the climax of taking people away to what was termed a "home" was, of course, a very great benefit to society. He thought the most depressing visit he had ever paid to any institution was one which he had paid to a "home" in Lancashire for mental defectives; and yet, although it was so depressing, when one realized the amount of care and medical attention which was being given to those unfortunates and the sympathy of those who were instructing them, they did realize that something useful could be made of those young persons. Anything of that kind, however, meant team work—it meant the cooperation of the person who alone was qualified to say whether the individual was physically fit and the person who could measure his intelligence and perhaps say something about his emotional life, and the social worker who could say something at first hand about the home.

Cooperation of the kind exemplified by the meeting that night would justify, if he might put it that way, many of the researches that had been undertaken by men working in the psychological field. Very often, if there were a chance of cooperation between the medical adviser and the social worker, the views of the psychologist were not able to be brought out in their full significance. The joint cooperation of all three points of view must be invaluable and, although that was only a minor point of view, it must react upon those who were studying psychiatry and those who were teaching the thing, because it would make their knowledge of human nature and of the mind much more practical. It was only since psychiatry had been in the air and could point to certain achievements that public opinion had been invoked, had begun to wake up and think there was something in these psychological investigations. They suffered in Melbourne from not having a full department of psychology, but they could point to excellent work at the Teachers' Training College, notably by Dr. Cunningham and Dr. Bachelard who had done pioneer work which neither the Government nor the University had been able to extend nor acknowledge it as it should be. He hoped they would be able to do more and then the team work of the medical practitioner and those particularly skilled in the subjects of psychiatry and psychology with such social workers as magistrates and those doing settlement work and charity organization work, would make mental hygiene mean something not only to the community, but to those often pathetic individuals whom they were out to serve and whose lives they could make happier by making more useful.

MR. V. MCRAE said that he would like to say at the outset how much the Education Department appreciated the compliment that had been paid it when its representative was asked to speak. It was, of course, a matter which concerned the Education Department very intimately in many phases of its work. If, in spite of Professor Dawson, he might be permitted to speak of mental hygiene as a movement, he would say that the movement had from the very beginning the cordial and unqualified support of the Education Department. It was appropriate for more reasons than one that the meeting should have been held at that particular time. Reference had already been made to the fact that on Monday of the following week there began in Washington the first World Conference on Mental Hygiene. That evening was almost, if not quite, the anniversary of the formation of the first Council of Mental Hygiene in 1906 in Newhaven, United States of America, so that in setting about the formation of a Council for Mental Hygiene, Victoria had but come into line with a movement that had already had very successful results in other progressive communities.

He was very glad to hear Professor Dawson strike an optimistic note regarding the possibilities of doing something for the lesser grade of mental defectives, especially amongst children. He had heard it said from the public platform in Melbourne that in establishing special schools for mental defectives the Education Department was perhaps doing social harm rather than rendering a social benefit, as by educating those defectives they were rendering them more potentially dangerous to the community. He had never subscribed to that view; in fact for some years he had been regarded as a hopeless optimist in this matter of training and caring for mentally defective children. His own experience justified this view and he was very glad to have an assurance from such an authority as Professor Dawson that, in his experience, the fatalistic view of the future of mentally incapable children was not justified by facts.

The Education Department had for many years been endeavouring to do something for children suffering from mental defects and they had found that if the right classification could only be obtained early enough, it meant a very great difference to the child's future development and progress. All those present at the meeting would realize and did not need to be told that the greatest danger that children who were not intellectually alive and alert as their fellows, ran, was that they might develop what the psycho-analysts called the inferiority complex and their more fortunate schoolfellows certainly did their best to assist in the formation of that complex. The best that could be done for children of that type was to give them a new sense of self-respect and a new feeling of confidence in their own ability to do something that was worth doing, and in their own ability to make good in their particular environment. By getting these children properly sorted out early enough and having them taught in special classes or special schools for the graver kinds of defect and by having a modified programme in which there was less emphasis than usual on the more academic subjects and more emphasis upon such activities as hand work, singing, rhythmic and physical exercises of different kinds, these children could be led to develop a greater feeling of confidence in their own powers and ability to do something worth while.

He was glad to say that the formation of the Council for Mental Hygiene had the cordial and unqualified blessing of the Education Department which would be glad to help in any way in which their assistance might be deemed of value.

DR. VERA SCANTLEBURY said that there had been many movements in Victoria already that had dealt with mental welfare from many aspects, and she thought that the proposed council would help to coordinate such movements and give them an opportunity of consulting together. They wanted to prevent mental troubles and there was no doubt from what psychologists told them that they could prevent mental troubles to as large an extent as they could prevent physical ills by proper preventive educational measures. The welfare movement had tried to prevent disease by proper attention to diet and to general bodily hygiene which could not be separated from mental hygiene.

As Professor Dawson had pointed out, the work in the Tresillian baby homes was the same work as that being done throughout the State. They tried to educate the mother and those dealing with the child not to overstimulate nor overfatigue and overburden the child. They then attended to the nutrition of the child. Some of the authorities they had most to do with, had also been authorities in mental diseases and these authorities had stated that nutritional work would do a great deal to prevent a number of mental disorders and that the earlier nutritional work was applied, the better would be the prevention. They must tackle this work in the first six years of life. They must look upon this not only as preventive work and curative work, but as creative and constructional work and try so to organize the play of the child that it was constructive and give it occupations which would develop it and lead it to independence and efficiency instead of dependence and maladjustment, as was too often the case.

SIR STANLEY ARGYLE said that his view was that of a politician who had realized for some years the necessity

for work in that direction, but had failed miserably in giving effect to any schemes that had been brought forward. Several things had made him think that night that a meeting of such a body as the proposed council, might be extremely useful in the political world by bringing pressure on the authorities to do something. He would point out that some of the foundations had been laid, but public opinion had not been sufficiently emphasized to enable the superstructure to be erected upon the foundations. One speaker had referred to the urgent need of a residential school for mental defectives. There was such a school in the course of construction; no less a sum than £20,000 had been spent upon it in the last three years, yet it still remained idle and empty, unfinished, nothing done. Of course the financial problem was a serious one and that made it extremely difficult for governments and other responsible bodies to do anything, but he thought that the proposed council could very easily draw attention to the fact that an enormous amount of money had been spent by the Government in Victoria in maintaining in institutions people who ought not to be there. He had drawn attention in Parliament to the fact that for year after year they had gone on putting individuals into penitentiary institutions and asylums, the cost of their maintenance to the State amounting to enormous sums of money and they not only got nothing for it, but were actually drifting backwards, because some of those people were increasing for reasons which he need not go into, but were obvious to those who had studied the question. Such things should be brought before the public by the proposed council by deputations and by persistent reiteration that something must be done in the public interest and he was pleased and gratified to think that they had at last made a beginning by the formation of a body whose business it was to do the things which he had advocated.

MR. FRANK TATE said that he was present at that meeting as a visitor, but was entirely in sympathy with its objects and with the sentiments expressed by the previous speakers. What he liked about the proposal was that it advocated team work between men and women who had been serving the community in their various spheres and who were all touching different aspects of the same question. The formation of an association such as had been suggested, would be an admirable way to bring together the results of the work that was being done and would stimulate the doing of much that yet remained to be done.

He moved:

> That this meeting constitute itself as the Victorian Council for Mental Hygiene and that application be made for affiliation with the National Council for Mental Hygiene in Great Britain.

SIR RICHARD STAWELL seconded the motion which was carried unanimously.

DR. J. F. WILLIAMS moved:

> 1. That a full member of the Council should pay an annual subscription of ten shillings and should be entitled to vote.
>
> 2. That an associate member should pay an annual subscription of five shillings, but should not be entitled to vote.
>
> 3. That, subsequent to the meeting, full members should only be admitted on being proposed and seconded by full members of the Council and approved at a meeting of the General Committee.

DR. K. S. CUNNINGHAM seconded the motion which was carried unanimously.

SIR RICHARD STAWELL stated that His Excellency the Governor, Lord Somers, had graciously consented to act as Patron of the Council which had just been formed.

On the motion of MISS GUTTERIDGE, seconded by DR. M. A. BUNTINE, the following office-bearers were elected.

President: Sir Richard Stawell.

Vice-Presidents: Sir Henry Maudsley, Dr. W. J. Springthorpe, F. Tate, Esquire, L. A. Adamson, Esquire, Dr. Edward Robertson, Sir Stanley Argyle, Dr. Ernest Jones, P. Hanson, Esquire, Dr. S. V. Sewell, Miss Gilman Jones.

Honorary Treasurer: Sir William McBeath.

Honorary Secretary: Dr. H. F. Maudsley.

Honorary Assistant Secretaries: Dr. J. F. Williams, Dr. Guy Springthorpe.

DR. H. F. MAUDSLEY said that he would have much pleasure in formally proposing that the following persons be elected as General Committee of the Council. They would notice that the list proposed consisted of about sixty names, as they wanted the membership to be not less than thirty nor more than eighty. They did not want to make the Committee absurdly large, but the scope of the movement was so broad that they felt they had to take in a great number. He therefore formally proposed the following motion which was seconded by DR. ERNEST JONES:

> A General Committee be appointed to carry out the work of the Council, and to consist of the President, Vice-Presidents, Secretary, Assistant Secretary and Treasurer, besides other members elected from the Council, which members shall number not less than thirty and not more than eighty. The General Committee shall have power to co-opt up to this number from members of the Council. A third of this Committee shall retire annually and be eligible for re-election.

The names of the proposed General Committee were as follows:

Mr. J. Akeroyd, Mr. S. Addison, Professor Agar, Dr. N. A. Albiston, Dr. Margaret Anderson, Dr. M. P. Bachelard, Mrs. T. a'Beckett, Mr. D. Black, Dr. Booth, Mr. G. S. Browne, Dr. M. A. Buntine, Dr. Caterinich, Professor Copeland, Mr. C. R. Croll, Dr. K. S. Cunningham, Mr. J. R. Darling, Dr. J. Dale, Dr. P. Dane, Dr. A. P. Derham, Dr. R. S. Ellery, Dr. D. Embelton, Dr. Eileen Fitzgerald, Mr. Foster, Dr. M. Gamble, Miss Glass, Dr. C. G. Godfrey, Dr. Boyd-Graham, Dr. J. Grieve, Professor Gunn, Miss Gutteridge, Mr. R. N. S. Good, Dr. Green, Mr. Hughes, Professor Wood-Jones, Reverend W. D. Jackson, Mr. Kelly, Mr. Kennedy, Archdeacon Lamble, Dr. L. S. Latham, Mr. Lawton, Dr. T. F. Mackeddie, Mr. J. McRae, Miss Macintyre, Dr. Kate MacKay, Dr. McPhee, Dr. H. F. Maudsley, Dr. F. K. Morris, Professor Osborne, Miss Edith Onions, Dr. C. R. Player, Dr. G. Pennington, Dr. A. J. W. Philpott, Mrs. Skene, Dr. M. A. Schalit, Dr. Sebire, Miss Helen Strong, Dr. Frances Stevenson, Dr. Guy Springthorpe, Dr. Vera Scantlebury, Mr. F. Shann, Dr. Georgina Sweet, Dr. M. D. Silberberg, Mr. L. J. Whiteoak, Dr. J. F. Williams, Professor L. J. Wrigley.

The motion was carried unanimously.

DR. GUY SPRINGTHORPE moved:

> That this Council will give power at this meeting to the General Committee to draw up a Constitution and to appoint an Executive Committee, also to arrange for formation of various Sub-Committees to organize and carry out the work of the Council.
>
> Subject to the approval of and amendment by the General Committee the following Sub-Committees are suggested. Alterations in these or formations of further sub-committees may be considered at a later date by the General Committee following suggestions by members of the Council.
>
> Proposed Sub-Committees: 1. Educational. 2. Child guidance. 3. Vocational guidance. 4. Industrial hygiene. 5. Mental deficiency. 6. Early treatment, after-care of mental disorders. 7. Delinquency and crime. 8. Finance and publicity.

The motion was seconded by DR. M. A. SCHALIT and carried unanimously.

Correspondence.

THE ANNE MACKENZIE ORATION.

SIR: I have read with much interest the excellent Anne MacKenzie Oration delivered at Canberra by Dr. Frank S.

Hone who is to be congratulated on keeping up the high standard set in the first oration by Dr. Cumpston, Federal Director of Health.

Whilst agreeing with and actually willing to dot the i's and cross the t's of every word Dr. Hone said, I wish to draw attention to Sir Andrew Balfour's comments on a report over the sanitary reform in Western Australia in 1882:

> It is the same old story. What Western Australia needed was a good going epidemic, preferably of cholera, the great loosener of purse strings;

followed by Dr. Hone's remarks:

> that panic is a bad thing on which to rely; for many foolish things are done under its influence, as was witnessed in regard to masks and inhalation chambers in the influenza epidemic in 1919.

Speaking from experience of an outbreak of plague in 1908 and the serious typhoid fever outbreak in 1909 in Auckland, New Zealand, epidemics of typhoid fever in Tasmania in 1910-1912, the small pox scare in Sydney in 1913 and the ninth plague outbreak in 1922, I can testify to the impetus which is given to sanitary reform by an outbreak of epidemic disease.

However true it may still be, that as Ruskin says: "Any interference which tends to reform and protect the health of the masses is viewed as unwarranted interference with their vested right to inevitable disease and death"; yet one has noticed of late years, largely owing to the great amount of propaganda in the press, from health weeks and by voluntary organizations, that the public are more willing to follow advice, although such is still to many people the vice most detested.

With regard to the use of masks and inhalation chambers in the influenza pandemic in 1919, I personally refused to wear a mask unless it were made compulsory, when as an officer and a law-abiding citizen I was in duty bound to do so.

In season and out of season I advocated merely the nasal inhalation of a hypertonic supersaturated sea-salt solution, indicating the freedom of surf bathers from influenza, in addition to advocating a righteous, sober and well regulated life.

With regard to inhalation chambers, I had the gratification of getting these stopped when an employee of the Town Hall in charge of one of the chambers became violently sick from the use of zinc sulphate. Whilst the use of the portable steam inhalers by inspectors masked, in white coats, almost reminiscent of the days of the Spanish Inquisition, so scared certain people when they were first used, that the absurdity of their use in view of the adverse psychological effect was so apparent that they were speedily consigned to the scrap heap.

One has to remember, however, that in times of a serious epidemic, especially in a country like Australia with such a large press, the public clamour for something to be done and there is always the risk of doing too much or overdoing some little thing which at the time someone thinks is a panacea.

As far as the general public is concerned, it is unfortunate that such an admirable address on the human factor in environment has not been reproduced *in extenso* in the lay press, as its educative effect would be valuable.

Yours, etc.,

J. S. PURDY,

Metropolitan Medical Officer of Health.

Queen Victoria Building,
Sydney.
May 28, 1930.

TONSILLECTOMY.

SIR: Dr. H. Gray asks a question and then makes an assertion which is not correct. He could not know of cases of serious bleeding from reverse guillotine in my practice. First, because I never had any until 1924 at the Children's and, secondly, because he left Melbourne Hospital before I used it.

Apart from theory, my statement concerning the crushing cutting guillotine was based on the fact of the very large number of serious secondary hæmorrhages that occurred during the nine years that it was used by another surgeon.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne.
May 24, 1930.

UNDULANT FEVER IN AUSTRALIA.

SIR: The first case of undulant fever definitely diagnosed in Australia is recorded in the *Australasian Medical Gazette* of January 20, 1908.

It was under my care in the Sydney Hospital. Being puzzled over the symptoms, I consulted Dr. Tidswell, then Acting President of the Board of Health. He found that Dr. Ashburton Thompson had among many specimens of microorganisms which he had obtained from abroad, one consisting of Malta fever organism, *Micrococcus melitensis* (Bruce).

It was fortunately still alive. For fuller details I would ask you to refer to the number of the gazette in question.

Yours, etc.,

T. STORIE DIXSON.

215, Macquarie Street,
Sydney.
June 3, 1930.

Obituary.

ARTHUR STYLES VALLACK.

WE regret to announce the death of Dr. Arthur Styles Vallack which occurred at Sydney on June 7, 1930.

ROBERT LIONEL FAITHFULL.

WE regret to announce the death of Dr. Robert Lionel Faithfull which occurred at Moss Vale, New South Wales, on June 8, 1930.

Books Received.

ANATOMY, DESCRIPTIVE AND APPLIED, by Henry Gray, F.R.S., F.R.C.S.; Twenty-fourth Edition; Edited by T. B. Johnston, M.B., Ch.B.; 1930. London: Longmans, Green and Company Limited. Royal 8vo., pp. 1482, with 1301 illustrations, of which 607 are coloured.

VARICOSE VEINS, WITH SPECIAL REFERENCE TO THE INJECTION TREATMENT, by H. O. McPheeters, M.D., F.A.C.S.; Second Edition, revised and enlarged; 1930. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 233, illustrated with half-tone and line engravings. Price: $3.50 net.

MODERN OTOLOGY, by Joseph Clarence Keeler, M.D., F.A.C.S.; 1930. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 865, with 90 illustrations and 15 coloured plates. Price: $10.00 net.

ELEMENTARY PRACTICAL BIOCHEMISTRY, by W. A. Osborne and W. J. Young; Third Edition; 1930. Melbourne: W. Ramsay. Demy 8vo., pp. 154.

DISEASES TRANSMITTED FROM ANIMALS TO MAN, by Thomas G. Hull; 1930. London: Baillière, Tindall and Cox. Royal 8vo., pp. 350, with illustrations. Price: 25s. net.

NOT SO QUIET, by Helen Zenna Smith, 1930. London: Albert E. Marriott Ltd. Crown 8vo., pp. 240. Price: 6s. net.

PRACTICAL MEDICINE SERIES: PEDIATRICS, Edited by Isaac A. Abt, M.B., 1929. Chicago: The Year Book Publishers. Crown 8vo., pp. 448. Price: $2.25 net.

PRACTICAL MEDICINE SERIES: OBSTETRICS, Edited by J. B. De Lee, A.M., M.D., and J. P. Greenhill, B.S., M.D., F.A.C.S. GYNÆCOLOGY, Edited by J. O. Polak, M.D., 1929. Chicago: The Year Book Publishers. Crown 8vo., pp. 660. Price: $2.50 net.

NORMAL FACTS IN DIAGNOSIS, by M. Coleman Harris, M.D., and Benjamin Finesilver, M.D., 1930. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 247, illustrated with forty-two engravings, some in colours. Price: $2.50 net.

A TEXTBOOK ON THE NURSING AND DISEASES OF SICK CHILDREN FOR NURSES AND WELFARE WORKERS, by Various Authors, Edited by Alan Moncrieff, M.D., B.S., M.R.C.P., 1930. London: H. K. Lewis and Company Limited. Demy 8vo., pp. 596, with 111 illustrations. Price: 15s. net.

A MANUAL OF DISEASES OF THE EYE, by Charles H. May, M.D., and Claud Worth, F.R.C.S.; Sixth Edition, 1930. London: Baillière, Tindall and Cox. Demy 8vo., pp. 483, with 22 coloured plates. Price: 15s. net.

VARICOSE VEINS, HÆMORRHOIDS, VARICOCELE, HYDROCELE AND THEIR TREATMENT BY INJECTION, by Ronald Thornhill, M.B., Ch.B., 1930. London: Baillière, Tindall and Cox. Crown 8vo., pp. 126. Price: 5s. net.

MEDICAL DIRECTORY issued by the National Medical Association of China on the occasion of the Eighth Biennial Conference, February 2 to 8, 1930, at Shanghai. Post 8vo., pp. 240.

THE BACTERIOPHAGE AND ITS CLINICAL APPLICATIONS, by F. D'Herele, 1929. London: Baillière, Tindall and Cox. Demy 8vo., pp. 262. Price: 18s. net.

Diary for the Month.

JUNE 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 18.—New South Wales Branch, B.M.A.: Section of Obstetrics and Gynæcology.
JUNE 18.—Queensland Branch, B.M.A.: Eye, Ear, Nose and Throat Section.
JUNE 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 25.—Victorian Branch, B.M.A.: Council.
JUNE 26.—New South Wales Branch, B.M.A.: Branch.
JUNE 26.—South Australian Branch, B.M.A.: Branch.
JUNE 27.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. J. M. Hair (B.M.A.) has been appointed Honorary Assistant Surgeon, Coast Hospital, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. C. W. Anderson (B.M.A.) has been appointed Medical Officer of Health by the Dalwallinu Road, Board, Western Australia.

* * *

Dr. C. T. W. Upton (B.M.A.) has been reappointed Honorary Dermatologist (Visiting) to the "Mareeba" Babies' Hospital, South Australia.

* * *

Dr. N. R. Bennett (B.M.A.) has been reappointed Honorary Medical Officer at the Port Lincoln Hospital, South Australia.

* * *

Dr. H. L. Ashton-Shorter (B.M.A.) has been appointed Government Medical Officer at Kingaroy, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xviii.

ALFRED HOSPITAL, VICTORIA: Resident Assistant Pathologist.
AUSTIN HOSPITAL FOR CHRONIC DISEASES, HEIDELBERG, VICTORIA: Junior Resident Medical Officer.
HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Senior Resident Medical Officer (1), Junior Resident Medical Officers (2).
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Urologist.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer, Temporary Honorary Assistant Anæsthetist and Relieving Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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